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Per 1519 e. 113

GUY'S HOSPITAL REPORTS.

EDITED BY
H. G. HOWSE, M.S.,
AND
FREDERICK TAYLOR, M.D.

Third Series.
VOL. XXV.



LONDON:
J. & A. CHURCHILL, NEW BURLINGTON STREET.

MDCCCLXXXI.

16

PRINTED BY J. E. ADLARD, BARTHOLOMEW CLOSE, E.C.

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Annali Universali di Medicina e Chirurgia (per Dr. Corradi, Pavia, North Italy).
Transactions of the Brooklyn Anatomical and Surgical Society, 28, Madison Street, Brooklyn, New York, U.S.A.

ON PURPURA HÆMORRHAGICA, ACCOMPANYING THE GROWTH OF MULTIPLE SARCOMATA.

BY C. HILTON FAGGE, M.D.

AMONG the various causes of a purpura attended with hæmorrhage from mucous surfaces—the affection commonly known on the Continent as the Morbus maculosus Werlhofii, in memory of Werlhof, who was physician to the King of Great Britain in Hanover in the middle of the last century—there is one which seems to have been left almost unmentioned by writers. It is the rapid development of sarcomatous growths in various organs, or throughout the body generally. Several cases in point have occurred at the hospital within the last twenty years, and I believe that they are well worthy of being collected together and placed on record; and the more so, as they have been characterised by some other symptoms, the recognition of which might probably render a diagnosis of the real nature of such an affection more easy in the future than it has been in the past.

CASE 1.—*Purpura hæmorrhagica, preceded by symptoms as of rheumatism; sarcomatous tumours of skin, kidneys, and pericardium, secondary to a latent sarcoma of the ileum.*

(From the Report of Mr. H. H. STURGE.)

R. B—, æt. 25, was admitted into Guy's Hospital, on July
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28th, 1879, under Dr. Pye-Smith. He was a labouring engineer, married, but without children. His father, he thought, had died of rheumatic fever.

On Whit Monday (June 2nd) he got wet through. From that time he complained of occasional pains in the chest, over the outer side of the hips, and over the back of the left shoulder, but they were not bad enough to compel him to give up work.

On July 19th, however, he felt, on rising in the morning, severe pains in both shoulders and in both hips. He went to work, but came home at midday, and went to bed. The parts first attacked remained painful up to the time of his admission; but, in addition, he had pain between the shoulders, and stiffness and pain in the right side of the neck. During mastication there was pain over the rami of the jaws. Both knees became painful, but not swollen.

On the 23rd the left eye watered, and became almost closed by swelling; the right eye also ached, and was sensitive to light. He sweated profusely day and night, so that his shirts were "wringing wet." He felt hot, and "got out of bed for coolness." He had pain all through his head, both front and back; he was also giddy. He was able to walk for about six minutes on his way to the hospital.

He appeared pale, languid, and ill, though fairly nourished. In addition to the pains in the parts already mentioned there were three very tender spots on the chest, one to the left of the sternum, another below the right nipple, the third near the inner end of the left clavicle. The slightest pressure over the left iliac crest caused pain, which went half way down the thigh. There was no pain on pressing the hips laterally, nor on pushing upwards the soles of the feet. Scarcely any pain in the right hip and knee was caused by free movements of the limb, but over the anterior half of the right iliac crest there was great tenderness. There was also much pain round the right groin and in the tuber ischii. The upper limbs could be moved pretty freely, but raising the arms upwards or across the chest gave pain, especially on the right side.

The skin was hot and moist; there was an unpleasant sour smell, especially when the clothes were raised. The appetite was very bad, but there was much thirst. The tongue was moist, pale, and flabby.

There was some dulness over the bases of both lungs, and at the right base the respiratory murmur was deficient. Resp. 32; temp. 102°; the urine, of sp. gr. 1026, was acid, without sediment. The heart sounds were healthy. The heart's apex was slightly outside the nipple.

It was supposed that he was in the later stage of an attack of rheumatic fever; and on the 30th a systolic apex murmur was discovered when he sat up.

On the 29th it is noted that he was sweating profusely, especially over the head. At 6 p.m. the temperature was 102·2°; the pulse was 96. He was lying with his knees flexed and raised, as he found that this gave him most relief. At 9 a.m. the sweating was most profuse; temp. 102·3°; pulse 96.

30th.—He only dozed one or twice in the night. Temp. 102°; pulse 100. Pain chiefly at vertex of head and in left tuber ischii. Urine 1028, not clear, two pints six ounces in twenty-four hours.

Of the further progress of the case there are unfortunately no detailed notes. But it is reported that he went on well until August 9th, when a purpuric rash came out on the chest and on the abdomen. He also had hæmaturia. His eyelids and his scrotum became greatly swollen. His conjunctivæ were ecchymosed.

Some of the purpuric spots were noticed to be raised, and it was thought that they were inflamed and tending to suppurate or to form small furunculi.

He rapidly became worse, and died on August 14th at 9.55 p.m.

I made an autopsy on the 15th, sixteen hours and a half after death. Decomposition was already rapidly advancing, the scrotum, the thighs, and the upper arms being emphysematous, so that air escaped from them when incised. The scattered spots of purpura still remained visible. They were seated chiefly on the side of the chest and abdomen, and about the groins. Some of them had the ordinary appearance, being flat, and red or purple in colour; but others were distinctly raised, forming indurated purple nodules; and yet others had a central pale elevation with a narrow ring of purple discoloration round it. They were all small, from half a centimètre to a centimètre in diameter. When cut into they looked exactly as

if they consisted of slightly swollen granular masses of adipose tissue, or were minute lymphatic glands, grey and fleshy. It was not until I had examined the viscera that I perceived them to be small growths, although the idea of a neoplasm had occurred to me as a possible solution of the case before I opened the body, when I merely looked at the cutaneous affection. The microscope subsequently showed that the minute round and irregular cells which made up the nodules had a curious tendency to infiltrate the small lobules of fat, the adipose vesicles being separated from one another, and enclosed each in a kind of capsule of new growth.

The lungs were healthy, but the anterior edge of the left pleura was slightly ecchymosed. The pericardium was extremely ecchymosed, and on its visceral layer there were some flat, white, milky-looking spots and patches of peculiar appearance, and probably early nodules of the new growth. The heart was very soft and flabby.

The liver and the spleen were healthy. The kidneys weighed $12\frac{1}{2}$ oz. together. Each of them contained many white or pinkish, succulent-looking, sarcomatous nodules. Many were very ill defined, and several of them in one kidney appeared to involve the pelvis of the organ. The bladder contained bloody urine.

So far no lesion had been discovered which could be regarded as the primary starting-point of sarcomatous infection. At length, however, it was found that the last two inches of the ileum, including the ileo-cæcal valve, were greatly thickened, forming a massive tumour. This consisted of a fine homogeneous yellow material, yielding no juice on section. All the coats were infiltrated, the mucous membrane being thrown into smooth undulating elevations. The muscular coat was in part visible as a grey swollen line.

The growth everywhere consisted of small round or irregular cells, very thickly crowded together. In the intestine they were supported by a considerable quantity of well-developed fibrous tissue.

CASE 2.—*Rheumatoid symptoms lasting a month; purpura, hæmaturia, epistaxis, bleeding from gums, fatal in seven*

days from the commencement ; sarcoma of vesicula seminalis, neck of bladder, kidneys, omental glands.

(From the Report of Mr. ROBERT J. WAINSWRIGHT.)

F. J. A.—, æt. 38, was admitted, under Dr. Moxon, into Guy's Hospital, on May 9th, 1877. He was a cabdriver, and had always been a strong man up to five weeks before his admission. His mother died of hæmorrhage during a confinement. His father was alive and healthy. Six brothers and one sister were alive and well ; none had shown a tendency to hæmorrhage. He had been married fourteen years, and had four children living, two others having died of croup. About fourteen years back he had had gonorrhœa, which was not followed by stricture. He had never had gout nor rheumatic fever. Since an attack of measles he had been deaf, and had had a discharge from the right ear. His nose had bled sometimes, but very seldom, and never to any extent. He had always had plenty of green vegetables, fruit, and fresh meat.

Five weeks back he believes that he caught cold from sitting on a wet seat and being exposed to draughts. He suffered from pains in the back, legs, arms, and chest, chiefly in the joints ; the pains were felt first in one joint, then in another. His water was high coloured and became thick on standing ; he sometimes passed red gravel, which gave him pain. At night he was restless and hot and sweated profusely, the sweat having a very sour smell.

He was, however, able to do his usual work until six days before his admission, except at one time, when he remained indoors for a few days but not in bed. But on May 3rd he sent for a medical man. The following night, while in bed, he was attacked with severe pain, which lasted for several hours, in the pubes and round to the back. He applied a mustard plaster, and took some pepper in water ; violent retching and vomiting set in. Next morning he noticed, for the first time, purpuric spots on the neck, groins, and legs ; and when he passed water, which he did with difficulty, there was blood in it.

After this he remained almost constantly in bed. His water always contained blood, and sometimes looked like pure blood. He had much difficulty and pain in voiding it, and sometimes passed clots. There also were uric acid crystals. On May 5th

he had profuse epistaxis for about an hour ; he thinks he lost a pint and a half of blood. His gums have been sore, and blood has been continually escaping from them into his mouth.

The medical man who had attended him confirmed this report, and added that his temperature had ranged at about 100° and upwards.

He was a fairly-nourished man, much blanched, and very weak. His pupils were slightly dilated, and his eyes so sensitive to light that he was continually closing them. His mind was perfectly clear. There were numerous petechiæ on both legs and feet, on the arms (especially at the elbows), and over the front of the neck ; and there were a few on the chest. The tongue was clean and healthy-looking. The heart-sounds were faintly audible ; the impulse was imperceptible. The lungs appeared to be healthy, but it was not possible to examine the back as his nose began to bleed. The liver and the spleen seemed to be of normal size. The urine was of sp. gr. 1025, dark purple, very thick, not coagulating spontaneously, but turning nearly solid when boiled. The microscope showed in it an immense number of red discs, some leucocytes, no casts. There was slight tenderness over the bladder, not elsewhere in the abdomen.

At 1 p.m. the temperature was 102°, the pulse 160, the respirations 26.

The epistaxis continued from 12.15 p.m. all the afternoon, with a few short intermissions ; every now and then he would spit large clots from the back of the mouth. The blood did not come in large quantities at once, but trickled out of the nose down on the face, and backwards into the throat. He was obliged to make water rather frequently ; it gave him some pain, and he had to wait for some time before any passed ; the urine looked like pure blood, it contained no clots. At 4.45 p.m. the temperature was 101.6°, the pulse 136, very soft and compressible, respirations 25. There was a cold clammy sweat over the skin. He was able to answer questions without difficulty.

The diagnosis was "*purpura hæmorrhagica*." He was ordered to take Acid. Gallic. gr. xx, Tr. Opii ℥xv, Syrup. Simpl. ʒj, Succ. Limon. ʒss, Aq. ad ʒj statim, post horas tres, postea omni 6tâ horâ. For diet he was to have milk, eggs and fluid meat ; but he took only iced milk, altogether about two pints and a half.

During the night the bleeding from the nose still continued, but in small quantity; he spat from the mouth several large clots. At 9 a.m. on May 10th he died. Before death his breathing became extremely slow and laboured. His mind seemed clear, but he was unable to speak. The pupils were moderately contracted. There was no convulsion.

An autopsy was made the same day by Dr. Goodhart.

The body was spare; numerous small petechiæ were visible on the limbs and trunk.

The chief seat of disease lay in the urinary organs. The kidneys were large, each weighing about 8 oz. They were pale and mottled on the surface, and they showed a number of rounded tumours, varying in size from mere dots up to one centimètre in diameter, white, elastic, almost juiceless. The larger of these tumours extended almost through the whole thickness of the cortex, their margins were tolerably well defined, but they gradually merged into the kidney texture, as if infiltrating it. Many of them were ecchymosed in the centre. The pelves of the kidneys were healthy, except that on one side there was a considerable extravasation of blood beneath the mucous membrane.

The ureters were healthy; the right one contained some milky fluid, which, on examination, showed epithelioid cells. The bladder was distended, but its lining was normal, except that it was slightly ecchymosed. The veru-montanum and the lining of the prostatic urethra had a peculiar white rugose appearance, and their submucous tissue was much thickened, and evidently infiltrated by a growth. The prostate itself did not appear to be much diseased, but the right vesicula seminalis and the vas deferens were converted into a large mass of white firm growth, with their channels still persisting in its centre. The thickening of the vas deferens continued for some distance along it. The left vesicula was a little thickened, and was probably in an early stage of the disease. The testes were healthy.

It thus seemed that the primary growth was in the right vesicula seminalis and in the adjacent structures. Microscopically Dr. Goodhart found this to consist of a highly nucleated small-cell-infiltration of the mucous and submucous tissues.

After being hardened the kidneys showed the same kind of nuclear growth.

The only other part in which any new growth was found was the omentum, which was studded over with small flaky ecchymosed glands. The tonsils were large, but this appeared to be an ordinary chronic hypertrophy. The cervical glands on the left side were slightly swollen and fleshy. The spleen weighed about 10 oz.; it was soft. The medulla of the bones was normal. The brain, the lungs, the heart, the liver, and the intestines were healthy.

The pericardium was studded with small ecchymoses, and there were a few on the pleuræ. The lining of the stomach was covered all over with minute hæmorrhagic spots, and it contained a brown material—probably blood altered by the astringent medicine which had been given.

The blood throughout the body was thin, and looked like milk-chocolate. Yet Dr. Goodhart found no marked microscopic change in it. The white corpuscles were not in excess and were small. The red discs were of all sizes; there were many fine granules in the serum.

CASE 3.—Purpura and hæmoptysis fifteen days before death; hæmaturia four days later; hæmorrhage from gums; rapid swelling of the cervical glands and tonsils; fatal œdema of the larynx; lympho-sarcoma of cervical glands, tonsils, thymus (?), and kidneys (?).

Thomas C—, æt. 30, was admitted into Stephen Ward, under my care, on March 23rd, 1867. He was a hawker of fish, and was accustomed to lift heavy weights. He was married, and temperate in his habits; he had never had syphilis. He had had good health until February, when he began to have a cough, and to expectorate phlegm streaked with blood. From this he recovered, and returned to work until a fortnight before his admission. He was then suddenly seized with a dull heavy pain in his chest, which compelled him to give up his occupation. The night before his attack he had had pork and greens for supper, as was not unusual.

Six days later, on March 16th, a number of red and purple spots came out on his skin, and on the same day he spat a

quantity of dark blood, without coughing. Two days before admission, on the 21st, his urine became red or nearly black in colour, and it continued to be so afterwards. For about a week he had had pain in his testicles.

He was a well-nourished man, perhaps slightly yellow in complexion. There were small purpuric spots on various parts of the body, especially round the neck and over the præcordial region. There were also some on the gums, and one or two on the inner side of the cheek on the left side. The gums were not sore, but were perhaps slightly swollen. The lower right central incisor and the anterior bicuspid teeth were somewhat loose; and so, perhaps, were other teeth also. He expectorated a considerable quantity of blood, which seemed to come from the mouth, being mixed with saliva and mucous secretion. There was no evidence of pulmonary hæmorrhage. The heart-sounds were normal. Pulse 90; temp. 99·4°. The left lobe of the liver appeared to be enlarged, reaching down half way between the ensiform cartilage and the umbilicus. The spleen was much increased in size, its area of dulness extending for six inches vertically, and its edge being felt about half an inch below the ribs. The urine was of a dark red colour, containing red discs in abundance.

I ordered him Succ. Limonis ʒj, 4tis horis, and Tinct. Ferri Perchlor. ℥xv, ex Aq. ʒj, 6tis horis, and ice to suck.

On the 24th it is noted that he had passed four pints of urine in the last twenty-four hours; it was very black in colour, containing leucocytes as well as red discs. The leucocytes appeared not to be in excess. The bowels had not been relieved for two days, and therefore he took ʒss of castor oil on the 25th, which acted without causing any discharge of blood. There was slight hæmorrhage from the nares, but the urine became paler, and on the 28th it was free from blood. It was now turbid, but cleared when boiled. He still had hæmorrhage at times from the gums, but less than when he was admitted. There was a considerable effusion of blood beneath the left conjunctiva. He spoke of himself as feeling better. His appetite was good. He slept well at night. The temperature remained at from 99·4° to 99·9°. The pulse was found to be 130 on the 28th, but fell next day to 96. On the night of the 28th he had an attack of epistaxis, accompanied by headache.

On the 30th the report says :—" Last night he had profuse perspiration, and this morning at about 7 a.m. he noticed that the glands of his neck were enlarged. They are very tender to pressure. All the other lymphatic glands of the body are also swollen, though not so tender. He is very prostrate, and has scarcely power to move his head. He is perspiring freely. Pulse 120; resp. 24; temp. 103°."

On the morning of April 1st his most distressing symptom was found to be extreme dyspnœa. He was sitting up in bed, the supra-sternal and the supra-clavicular spaces being drawn in during every inspiratory effort. About noon, happening to be in the ward, I was startled by hearing him suddenly breathe very noisily. He became greatly distressed, very livid, and slightly convulsed. Rather suddenly the breathing ceased, and he fell back insensible. Galvanism was used, but, although it excited movements of the arms and neck, it caused no efforts at breathing. Tracheotomy was then at once had recourse to, a flickering pulse at the wrist being still perceptible. But this also proved to be of no avail.

The autopsy was made on the following day by Dr. Moxon.

The cause of death was œdema of the entrance of the larynx, especially on the left side.

The most important lesions were found in the lymphatic glands, the tonsils, the thymus, and the kidneys.

The cervical glands were much enlarged, measuring one inch in their long diameter; they were soft, of a pinkish cream colour, and blotched with ecchymosed patches. The axillary glands were but slightly smaller than the cervical, especially on the right side. The condition of the mediastinal glands is not noted. The glands at the portal fissure and those about the head of the pancreas were considerably larger than natural; the mesenteric, the lumbar, and the iliac glands were little, if at all, enlarged.

The tonsils projected as lobulated masses, half an inch in thickness. On section they presented the same appearance as the cervical glands, and so did the thymus, which formed a large pear-shaped mass.

The kidneys weighed 14 oz.; they were very pale, and they were spotted all over with white patches, which Dr. Moxon regarded as suppurating, but which it is perhaps allowable to

suspect of having been more or less diffused, multiple sarcomatous growths. Between the tubules small round cells were infiltrated, either within the capillaries or at least in the interstitial tissue. The pelvis (? of each kidney) was uniformly reddened and swollen, and this condition extended down the right ureter as far as the bladder. The urethra was healthy.

The spleen weighed 20 oz. ; its substance was pale and rather soft. It showed a few ill-defined patches of still paler colour, and the Malpighian corpuscles were here and there visible in it.

The liver weighed 128 oz. ; its tissue looked sodden and very pale, and there appeared to be an excess of fat in the cells at the periphery of the lobules. The microscope revealed the presence of large numbers of leucocytes, forming an interlacing network between the hepatic cells.

Within the capillaries of the substance of the heart also a very unusual number of leucocytes were seen ; Dr. Moxon counted twenty-five in a single short tract of vessel. I may observe, however, that I had examined the blood microscopically a day or two before death, and had failed to detect leukhæmia. There was certainly at that time no large excess of white corpuscles.

The pleuræ and the pericardium were much ecchymosed, and the pia mater and the peritoneum were slightly so. The coats of the stomach and intestines also showed very many small patches of extravasated blood.

There was early tubercular disease at the apex of each lung, consisting of cheesy and partly calcified masses, as well as many tubercles. (Probably this was the cause of the pulmonary symptoms which had existed about six weeks before his death.)

CASE 4.—*Rheumatoid pains three weeks before death ; spongy and bleeding gums ; anæmia ; fever ; purpura ; enlarged thymus ; encephaloid growth in mesentery.*

(From the Report of Mr. H. ASHBY.)

Alfred K—, æt. 28, a coachman, was admitted, under Dr. Wilks, into Stephen Ward, on April 17th, 1872. He said that for some weeks he had had headache and neuralgia. His gums had been very spongy, and had bled a great deal. His teeth had been loose and painful, and he had had severe pains running

up to the head. Ten days before admission he had pains in his joints and took to his bed. Ever since he had had severe pains in the elbows, knees, and shoulders, which, however, seemed to be diminishing in intensity. Both his father and his mother were alive and subject to rheumatism. He himself had never had gonorrhœa nor syphilis.

He was an anæmic-looking man; his tongue was brown, his breath was foul, his teeth and gums caked with blood. Pulse 128; temp. $100\cdot2^{\circ}$; resp. 28. The heart sounds were normal. He complained of aching pains all over his body, especially when his loins were pressed. The urine, of sp. gr. 1030, contained no albumen.

April 18th.—Pulse 108; temp. $100\cdot2^{\circ}$; resp. 26. He is in much the same condition, bleeding from the nose and gums; he still complains of pain and tenderness all over him. Ordered Mist. Quinæ ʒj, t. d.; Gargar. Aluminis; tannic acid to be applied to the gums and to the nose.

20th.—Pains still very bad, especially in the head, so that he cannot sleep. Appetite bad. Bowels relaxed two or three times a day.

25th.—Pulse 150; resp. 24. Raised purple spots have appeared on the abdomen, which do not fade on pressure. There is no tenderness of the abdomen.

26th.—Pulse 174; resp. 30; temp. $101\cdot8^{\circ}$. Some of the purpuric spots have faded to a dirty colour, other fresh ones are appearing. There are also some small clear vesicles over the body. The gums and the nose still bleed. Ordered brandy ʒiv; beef tea.

In the latter part of the day he fell into a semi-comatose condition, and died quietly the same evening.

I made a partial autopsy on the following day.

The brain was not examined.

Extending from the pericardium to the thyroid body there was an elongated firm mass, which appeared clearly to be an enlarged thymus. It was whitish and yielded but little juice on section.

The pericardium was ecchymosed.

In the mesentery was a large encephaloid mass, the size of a billiard ball. It contained numerous hæmorrhagic patches, and one large brown laminated clot exactly like that in an

aneurism. At one spot the growth reached the intestines, and there was an ulcer which tended to perforate the serous membrane. The mesenteric glands were widely affected with a similar growth, and also the subserous tissues generally. This was especially the case in the recto-vesical pouch, where there was a thick layer of whitish firm material.

The liver weighed 72 oz.; it was large and fatty, and along the portal canals there was some of the new growth, but in no considerable quantity.

The spleen was healthy.

The kidneys were mottled and apparently affected with Bright's disease.

The bladder was healthy; the prostate small.

CASE 5.—Purpura; spongy gums; multiple growths in and beneath skin, and in lymphatic glands.

Robert H—, aged about 25, a bank clerk, came to me in February, 1876. He said that about a fortnight before Christmas, 1875, he had got very cold, and for three days he was chilled through. He had never been well since. He had a cough, and his nose was stuffed up. His mouth and his tongue were stained with blood; his gums were slightly spongy. He had purpuric spots on the legs, seated round the hair-follicles; above the right ankle there was an effusion of blood of some size. About the chest there were indefinite stains, but these I noted to be "associated with distinct flat thickenings." Two enlarged glands could be felt at the back of the neck. The temperature was 101°, even in the morning.

It appeared that he was not accustomed to eat any vegetables, and therefore I at first regarded the case as one of scorbutus, and prescribed lemons, watercresses, and other fresh green food, and a mixture containing the tincture of acetate of iron. The subcutaneous tumours I thought to belong to an accidental molluscum fibrosum.

It very soon became apparent, however, that this diagnosis was a mistake. He rapidly became extremely anæmic, and lost all his muscular strength. The lymphatic glands in various parts of the body grew so large that the case was subsequently

regarded as one of that variety of Hodgkin's disease, which from a histological point of view, would be called a lymphosarcoma. The subcutaneous and the cutaneous growths also became very numerous. He repeatedly had hæmorrhage from the bowels, and once he nearly died in the water-closet, being found there in a fainting condition. I think there was troublesome epistaxis, but of this I am not sure, as I have no further notes of the case, having seen him only once or twice afterwards at his own house, in consultation with Mr. Llewellyn, of Whitechapel, who attended him. He died at the end of a few months. There was no post-mortem examination.

CASE 6.—Fatal illness lasting five weeks and a half; spongy gums; multiple tumours on scalp and body, one of which discharged blood; diagnosis at first scorbutus, afterwards melanosis; growths scattered in the viscera.

Susan G—, æt. 45, was admitted into Mary Ward, under Dr. Pavy, on March 13th, 1861. She was extremely ill, wasted, and very fallow. She said that she had been ailing for about three weeks. A lump was found on her head, which broke and discharged blood; this she attributed to a blow. There was also observed on the skin several lumps of a dark colour, and as her gums bled the case was at first regarded as one of scorbutus; subsequently the opinion was that the disease was melanosis. She presently had brain symptoms, sank into a comatose condition, and died on March 31st.

Dr. Wilks made the autopsy. The body was much wasted and of a yellowish colour. The mass on the scalp appeared to have contained fibrin as well as blood. On the body were a number of small swellings, the largest the size of a marble. These, when cut into, were found to be quite circumscribed, of a dark red colour, and apparently composed of fibrin; some, however, were very soft and contained much liquid blood.

On the surface of the brain there were four or five red spots, which looked as if made up of firm fibrin mixed with blood. At the extreme end of the left posterior lobe was a mass, of the size of a walnut, with hæmorrhage and yellow softening of the brain substance around it.

Each lung contained twenty or thirty firm dark red masses ; one, in the left lower lobe, was of the size of a closed fist. Similar growths existed in the liver and in the spleen ; in the former organ there were two, each as large as the fist ; some were softening in the centre.

The heart and the kidneys were healthy.

The uterus was large ; in its interior was a soft membranous layer, like a decidua.

The cases recorded in this paper constitute a large proportion—if not an actual majority—of all the fatal examples of purpura hæmorrhagica which have occurred in the hospital during the last twenty years. Indeed, from what I have seen of the occasional difficulty of recognising diffused sarcomatous infiltrations of the bones and periosteum in the dead subject, unless careful search is made for them, I feel some hesitation as to whether such disease may not have been really present in some of the few cases which have been set down, even after an autopsy, as examples of Werlhof's *morbus maculosus*.

It is true that Case 2, and perhaps Case 5, come fairly within the category of Hodgkin's disease. But although it is well known that purpura and epistaxis and other hæmorrhages sometimes occur as complications of that disease, I am not aware of any instances hitherto recorded in which they have been among the earliest and most conspicuous symptoms. There is, indeed, a case related by Virchow, in the 'Deutsche Klinik' for 1859, and alluded to in vol. ii of the 'Krankhaften Geschwülste,' at p. 577, which (it is said), "unter dem Bilde einer Purpura verlaufen war." But it appears that the patient was admitted into the Charité Hospital moribund, and no details are given as to the duration or early symptoms of his illness.

One of the most interesting features of the sarcomatous disease is the rheumatoid affection by which in several of these cases it was ushered in. This, in one instance at least, was attended with very profuse sour sweats ; but it is noteworthy that none of the joints were found to be the seat of an effusion of fluid. I am sure that one cannot be too guarded in diagnosing rheumatic fever whenever one fails to make out definitely that the articu-

lations themselves are attacked. In this connection it is perhaps worth while to allude briefly to a case which occurred in Dr. Moxon's ward in 1876, of a man who died, after about three months' illness, of multiple sarcomatous growths in the skin and the subcutaneous tissue, as well as in the different viscera. In that instance there was neither purpura or hæmorrhage from the mucous membranes. The chief symptom was wasting, for which no cause could be found, until some nodules of new growth became perceptible in and beneath the integuments of the chest and limbs. On referring to the clinical report, I find, however, that the patient stated that his illness originally began, eight weeks before his admission, with pains in the shoulders, which, after three days, were so severe as to compel him to take to his bed. Presently the hips, the knees, and the ankles, all of them became painful, one after the other. Whether the articulations themselves were affected seems, however, to be doubtful, for the report goes on to say that the pain extended down the limbs; it occasionally passed off for a time; the perspiration was not noticed to have an acid smell. He complained of giddiness and faintness. When admitted, five weeks and a half before his death, his face and lips were described as having been anæmic; his countenance was anxious. The temperature was normal, and continued to be so.

It is worthy of notice that in at least three of the cases recorded in this paper the commencement of the disease was definitely attributed by the patient to a chill or to getting wet through. I know that many pathologists would maintain that a neoplasm cannot possibly have its origin in such a cause; but for my own part I must confess that I am not sure of it.

Different views may be taken with regard to the relations between sarcomatous growths and purpura. One is that a minute development of sarcomatous tissue, with vessels made up of embryonic cells, occurs at each spot which becomes the seat of an effusion of blood; or, perhaps, that sarcomatous cells, or nuclei, or even leucocytes in an abnormal condition, become lodged in the capillary vessels here and there, and produce softening of their walls after the manner of emboli. In support of such notions is the fact that in Case 1 some of the purpuric patches corresponded with obvious sarcoma

tous nodules. And in the brain it is well known that a sarcoma or a glioma may become the seat of profuse hæmorrhage, which may tear up its structure so that there is great difficulty in recognising it, and so that a careless pathologist may easily imagine the case to be a simple one of ordinary apoplexy. This point, indeed, must always be borne in mind whenever an effusion of blood is found in a young subject, or when it is seated in a part of the brain, such as the cerebellum or the cortex, in which there are no large arterial vessels.

And in 1877 I made an autopsy which showed that even in the muscles, and in the lungs, sarcomatous growths may very closely resemble simple effusions of blood. The case was that of a boy, *æt.* 15, who had been admitted under Mr. Cooper Forster for a fracture of the thigh; he had fallen while carrying two shutters, and they had struck on the limb and broken it. Union appeared to take place as usual, but subsequently it was found that the fractured ends of the bones were still separated. The thigh became very painful and greatly swollen. An incision was made, and a quantity of blood escaped. Amputation was performed, but he sank, and died in a few hours. I examined the parts and found them to be in a very remarkable condition. The original line of fracture could still be seen, the surfaces of the bones corresponding pretty well. Round the upper fragment was a thin shell of callus. All the parts were soaked in blood, so that at first it was very difficult to detect any further pathological change. But on making sections of the muscles I found that the reddish-black blood-stained appearance did not fade gradually at its margins, but was limited definitely by convex edges. There were also slight indications of a new growth in the medulla of the bone and about some loose fragments. In the lungs there were five or six scattered secondary nodules, of the size of marbles, which repeated in a striking manner the characters of the primary growth in the thigh. They were of a reddish colour, and projected above the level of the rest of the surface. But they felt quite soft; and when they were cut into a quantity of blood squirted out of them and they collapsed, leaving cavities surrounded only by a very narrow margin of indefinite-looking tissue, outside which, again, was healthy-looking lung-substance. Under the micro-

scope the presence of a neoplasm was not much more easy of determination than with the naked eye. At length, however, I discovered in the muscles of the thigh some masses of delicate spindle-cell tissue, with large oval nuclei, containing also cells of more irregular form. In the lungs some similar structures were made out; but the obvious margins of the broken-down secondary growths appeared to be nothing more than patches of a catarrhal pneumonia, the new tissue being in the centre of what looked like clot, filling the cavities.

But another view is to regard the purpura, the spongy state of the gums, and the epistaxis, as the joint results of a profound cachexia or alteration of the blood, analogous to that which is present in pernicious anæmia, in splenic leukæmia, and, indeed, in scorbutus itself. In no fewer than five of the six cases recorded in this paper there was hæmorrhage from the gums; and in one of them it is noted that the teeth were loosened. In splenic leukæmia a morbid state of the gingival tissues has been described by Mosler; it was present in a case of that disease which occurred in the hospital in 1878.

It would be natural to speak of a "sarcomatous infection" of the blood as accounting for the various symptoms under consideration; but such a way of stating the matter would be liable to misapprehension. Case 1 is, in fact, the only one in which a primary sarcomatous growth was discovered, from which all the other tumours had evidently had their origin. It is true that in some of the cases which occurred several years back the presence of such a growth was perhaps not sought for so carefully as we should now search for it. And it must be admitted that to prove the absence of a primary sarcoma in some one or other of the bones would require that an autopsy should be conducted in a manner which is altogether impracticable. Only a few weeks ago I was examining the body of a man who had died under my care of wasting, attended with severe pains in various parts of the body; and I found beneath the periosteum of a large number of the bones, and in their cancellous tissues, an abundant sarcomatous growth, which not only caused no tumours that could have been felt during life, but did not even raise the muscles or show any signs of its presence until the bones themselves were exposed. In that instance I could not say that any one tumour

was of older date than the others; but it is obviously quite conceivable that a single intra-osseous or subperiosteal sarcoma might in another case easily be altogether overlooked, the scattered secondary growths in the viscera being alone detected. Still, I must confess that I hold strongly to the belief that multiple sarcomata are often developed in the connective tissue throughout the body—just as a large number of nodules of molluscum fibrosum are found beneath the skin—altogether independently of any infective process. Those who adopt a different view seem to me often to have recourse to very unsatisfactory expedients; as when, for instance, they refer melanotic tumours scattered in the various organs to a source in some small pigmented mole, which may never, up to the time of the patient's death, have shown any tendency to take on an active growth, and may, in fact, never have attracted his notice. No one thinks of the growths in Hodgkin's disease as having always a starting-point and spreading by infection. Yet there are many cases of Hodgkin's disease in which definite tumour-masses are found bearing no relation in configuration to the tissues in which they lie; and I think it is altogether impossible to limit the definition of Hodgkin's disease to those cases in which the growth is histologically a lymphoma, or, indeed, to regard it as corresponding with any single kind of new growth. Conversely, it is noticeable that in some cases of sarcoma the growths preserve the exact shape of the internal organs, even where the increase in size is enormous. In 1875 I examined a case in which there was an immense sarcoma of an undescended testis, weighing eight pounds. Along its outer side there ran a distinct rim or ridge, which perfectly represented the epididymis. Recently I was inspecting the body of a girl, æt. 10, who died of a mass of sarcomatous glands in the neck, which were probably secondary to a large, firm, fleshy tumour in one side of the broad ligament of the uterus. The adjacent ovary was about twice the size of the opposite one; its substance was opaque white, and made up of round sarcomatous cells, yet it retained an absolutely normal shape. I do not think that a carcinoma or epithelioma ever thus adjusts itself to the configuration of the natural structures; and I am satisfied that (except in very rare cases, such as those of double primary cancer of the mammæ) those

kinds of tumour, when multiple, are always derived from a primary source.

One interesting point in the cases recorded in this communication is the fact that in two of them (the patients being men, ætat 28 and 30, respectively) the thymus was enlarged, and apparently affected with the growth. For in one case of Hodgkin's disease, which occurred in 1860 at the hospital, in a girl æt. 10, the thymus was also found of very unusual size. It was so likewise in the girl whom I have just mentioned as having died of sarcoma of the broad ligament, ovary, and cervical glands. But in that instance Mr. Symonds found on microscopical examination that the enlarged thymus was not itself sarcomatous, but possessed a normal structure. The only other disease in which I remember to have seen a persistent thymus in an adult is exophthalmic goitre. It was so in two out of six or more cases of this disease that have ended fatally at the hospital within the last twelve years; one patient was twenty-nine, the other was twenty-one years old. In one instance the organ was four inches long, and had a maximum thickness of three quarters of an inch.

THERMOMETRIC SCALES.

BY THOMAS STEVENSON, M.D.

NOTWITHSTANDING the almost general adoption of the thermometric scale of Celsius—commonly known as the centigrade scale—by scientific men in this country, and in France and Germany, the scale of Fahrenheit is still generally used in this country for meteorological purposes, and by the physician. That of Réaumur is but little known among us, though it is the scale commonly used over a large portion of Eastern and South-eastern Europe. In even the best and most complete of our English text-books the history of these scales is almost entirely ignored. An account of the origin of the three common thermometric scales may therefore be of interest to medical men, and none the less because the basis of Fahrenheit's scale is founded upon observations upon the temperature of the human body. I may add that I am in great part indebted for my information to 'The Operative Chemist,' by S. F. Gray, a once well-known book, written apparently more than half a century ago. The book is one now rarely met with, and this must be my excuse for so largely borrowing from, and even using the language of, that author.

FAHRENHEIT'S SCALE.

The thermometric scale of Fahrenheit was devised by him a little after the year 1714. In the 'Acta Eruditorum' of that

year it is stated that Fahrenheit had made two thermometers, to which were applied a scale, in which the difference of temperature between that of a mixture of ice and salt and that of the armpits or mouth of a healthy man (96°) was divided into twenty-four parts; to every four of which was appropriated a distinct name: 0 very great cold (supposed to be the absolute unit of cold); 4 ($=16^{\circ}$) great cold; 8 ($=32^{\circ}$) cold air; 12 ($=48^{\circ}$) temperate; 16 ($=64^{\circ}$) hot; 20 ($=80^{\circ}$) very hot; 24 ($=96^{\circ}$) insupportable heat. It is probable that Fahrenheit had formed to himself an idea of six equal gradations (each $=16^{\circ}$ F.) of temperature from his point of extreme cold to that of insupportable heat, but, six gradations being too few for precise observations, he was induced to divide them into quarters, and thus obtained twenty-four.

It is not known how soon afterwards he was led to divide each of these twenty-four gradations for still greater precision, but in a paper of Fahrenheit's, in the 'Philosophical Transactions' for 1724, it appears that his meteorological or spirit thermometers were graduated from three fixed points into 96° . Boerhaave states that the bulbs were blown of such a size as to contain so much alcohol as would fill 1933° of the scale, if it were produced so long.

The three fixed points were obtained from—(1) a mixture of ice with sal ammoniac, or common salt, which furnished the zero of the scale; (2) a mixture of ice and water, which gives 32° ; (3) the point obtained by holding the bulb (Fahrenheit made his thermometers small and his bulbs cylindrical) in the mouth or under the armpits of a healthy man, which gave his 96° , the highest limit of his original scale. This is the number of degrees into which the mathematician Bird divided the quadrant.

Amontons having discovered that water boils at a certain degree of temperature, and other observers that the height of the barometer was affected by the different temperature of the mercury in the tube, Fahrenheit constructed some mercurial thermometers to ascertain the temperature at which different liquids boil. According to Boerhaave, these thermometers had their bulbs blown of such a capacity as to contain as much mercury as would fill $11,124^{\circ}$ of the scale if prolonged so far; but the scale, after having its fixed points determined, as

already stated, was continued to 600° only, for at that point the mercury began to boil (mercury boils at 643° Fahr.). With these thermometers Fahrenheit found that rain water boiled at 212°.

At present the mercury thermometers with Fahrenheit's scale are graduated from two fixed points only—(1) the point of melting ice, which gives the 32°; (2) boiling water when the barometer stands at 29·905 inches at the latitude of London; the distance between these points is divided into 180°, and the scale continued up and down.

It is a remarkable coincidence that the expansion of mercury is very nearly $\frac{1}{10,000}$ th of its bulk, measured at 32° Fahr., for each increment of 1° Fahr. (one volume of mercury at 32° becomes 1·018153 at 212°).

RÉAUMUR'S SCALE.

Sir Isaac Newton is said to have been the first who conceived the idea of making the degrees of the thermometric scale aliquot parts of the liquid measured at the freezing point; and Réaumur put the idea into practice.

Réaumur made use of large thermometers, whereas, as has been just stated, Fahrenheit used the far preferable small instruments. Réaumur used bulbs four inches and a half in diameter, and their capacity would probably be about a pint and a half. The tubes were a quarter of an inch in diameter. To graduate them he used a pipette, which was filled with water to a mark and its contents delivered into the thermometer. The size of the pipette was such that it required to be filled 1000 times to fill the bulb of the thermometer and some part of the tube. Assuming the bulb of the thermometer to hold a pint and a half such a pipette would deliver about thirteen minims. The scale of the thermometer had a double graduation, one to the left, denoting the number of pipette-measures, the other to the right, numbered upwards and downwards, the zero being placed opposite to 1000 on the left-hand scale. The upward series denoted thousandths of dilatations, and the downward series thousandths of contractions. Thus:

1004	4° of dilatation.
1008	3° ,,
1002	2° ,,
1001	1° ,,
1000	0° zero.
999	1° of contraction.
998	2° ,,
997	3° ,,
996	4° ,,

For meteorological purposes Réaumur filled his thermometer with spirits of wine diluted with water, until he found by repeated trials that 1000 measures of it when ice cold expanded in the thermometer to 1080 when the thermometer was dipped into boiling water. Hence 80° of this, the real Réaumur's thermometer, was not the boiling point of water, but that of the diluted spirit which he used, and, according to Dr. Martine, corresponds to 180° Fahr., so that each degree of Réaumur's scale is equal to 1·85° Fahr.

The degrees of Réaumur's scale are usually converted into degrees of Fahrenheit's scale by considering each degree of Réaumur as equal to $2\frac{1}{4}$ ° of Fahrenheit.

It is stated that this difference in the conversion of the scales arose thus:—M. de Luc, the French reader to Queen Caroline, Consort of George III, divided the space between the freezing and the boiling points of water, as marked on his mercurial thermometers, into 80°. Réaumur, it will be remembered, divided the space between 32° and 180°, *i.e.* 148° Fahr. into 80° Réaumur. Condamine is said to have advised de Luc to change his 80 for some other number, on the ground that his scale would be confounded with Réaumur's. De Luc, however, considered 80 to be a convenient number, as it has several divisors, and declined to follow Condamine's advice. Thus, the two thermometric scales of Réaumur and de Luc, although so different, have been confounded. De Luc's scale is given in his '*Recherches sur les Modifications de l'Atmosphère*' in 1772, and is now always termed, though incorrectly, Réaumur's scale.

De Luc's scale (termed nowadays Réaumur's), being based on the graduations of a mercury thermometer, is reducible by

calculation to Fahrenheit's scale. The true Réaumur's scale, being based on the graduation of a spirit thermometer, was not comparable with Fahrenheit's, the expansion of spirit being less uniform than that of mercury.

CELSIUS' SCALE.

Celsius, in 1742, taking the melting point of ice and the boiling point of water as his fixed points, divided the distance between them into 100° , mercury being the liquid whose expansion was measured. This scale was adopted in France in the 19th century under the name of the centigrade scale.

Fahrenheit's is the oldest, the centigrade the most recent, scale of graduation.

The melting point of ice not being sensibly affected by variations in barometric pressure the zeroes of Réaumur's and the centigrade scales coincide with 32° Fahr. The 80° of Réaumur and the 100° of centigrade coincide, and they indicate the boiling point of water in latitude of Paris under a barometric pressure of 29.922 inches (760 millimètres). The temperature 212° Fahrenheit is not coincident with these, but indicates the boiling point of water in the latitude of London under a pressure of 29.905 inches. Allowance being made for the differences in latitude between Paris and London, and for the corresponding alteration in the effects of gravity, it is found that a barometric pressure of 29.922 inches in Paris is equivalent to one of 29.914 inches in London. An alteration in pressure from 29.905 to 29.914 inches raises the boiling point of water nearly one sixtieth of a degree Fahr. Strictly, the 100° centigrade corresponds to 212.015° Fahr., or 212° Fahr. corresponds to 99.01° centigrade.

DISTENSION OF THE FRONTAL SINUS.

BY CHARLES HIGGENS.

CASE 1.¹—George W—, æt. 32, admitted July 10th, 1877. Twenty-five years ago suffered from abscess at inner canthus of right eye (lachrymal sac?). The abscess formed during recovery from scarlet fever. Eighteen years ago, he received a blow on the forehead from a bar of iron, but was not much hurt. Two years later some pieces of diseased bone were removed from the inner angle of the orbit. Patient has been at sea for the last sixteen years, as steward and cook. Has enjoyed fairly good health, but was never very robust. Has had typhoid fever and smallpox; no venereal disease.

Since the removal of bone, sixteen years ago, he has had no trouble about the orbit until eight months back. He then noticed a small lump at the inner angle of the orbit, which he attributed to cold taken during night watches. He noticed that the lump varied in size, being always much smaller in the morning, after a good night's rest, than at other times. Soon after the lump appeared he began to experience pain in the forehead, described as a kind of stretching and bearing down. The pain was constant, and has continued up to the present time. The swelling has gradually increased, and is still increasing, but varies in size at different times, being always much smaller in the morning, after rest, than in the evening, when he has been about all day.

¹ Reported in 'Guy's Hosp. Rep.,' ser. iii, vol. xxiii, 1878.

On admission.—There is a tense fluctuating swelling situated at the inner angle of the orbit, above the position of the lachrymal sac. The skin over the swelling is normal. There is no displacement of the eyeball. An incision was made into the swelling, when a large quantity of opaque yellow and rather tenacious fluid escaped; indeed, the quantity was so large that there seemed to be no end to the flow. A probe introduced through the wound passed for two inches upwards and somewhat backwards, and its extremity could be freely moved about, showing that it had entered a considerable cavity. The large end of a Webber's sound (about the size of No. 5 urethral catheter) was passed with but little force through the thin septum which intervened between the floor of the cavity and the nasal fossa.

July 16th.—Incision healed. No refilling of cyst. Discharge escapes in considerable quantity into nose, and passes backwards into pharynx; it is in no way offensive and gives no inconvenience.

At the end of August, there was no return of the swelling; the discharge had ceased running into the nose for some time.

CASE 2.—William T—, æt. 13, admitted March 7th, 1879. Six months ago, first noticed swelling at the inner angle of the left orbit; about the same time the eye began to water. The swelling has gradually increased and is still getting larger; the increase has been continuous, and there has been no variation of the swelling in size at different times of the day. Never had any pain. No history of injury. Knows of no cause for commencement of tumour.

On admission.—Hard, rather irregular growth, projecting from inner margin of left orbit; it is attached to bone by a broad base, extending from inner extremity of upper margin of orbit almost to level of inner canthus; it passes backwards into the orbit. The eyeball is pushed outwards; its movements are perfect; sight normal, there is no diplopia; nostril quite free; no teeth missing. Tumour looked upon as an exostosis from the inner wall of the orbit.

March 10th.—Patient placed under the influence of an anæsthetic. Incision made over tumour; cutting forceps applied to growth crushed through it at once, there being only a thin

shell of bone ; a quantity of grey, opaque, tenacious mucus escaped. The finger, passed through opening made by forceps, entered a large cavity, which extended for some distance upwards and somewhat backwards. A drainage tube was passed through the floor of the cavity into the nose, and its upper end fixed by strapping to the forehead. Cavity to be syringed out daily with solution of carbolic acid, 1 to 40.

June 3rd.—Drainage tube removed.

16th.—Fistulous opening at inner angle of orbit ; discharge escapes into nose. No air escapes through fistula on forced expiration with the nostrils and mouth closed. Still considerable thickening of bone.

July 21st.—Fistula smaller, but otherwise the same.

October 6th, 1879.—Swelling much contracted, small fistulous opening admitting a probe, which passes backwards nearly two inches ; cavity quite small ; epiphora ; still some discharge into nose.

January 19th, 1880.—Thickening of nasal bone, nasal process of superior maxilla, and internal angle of frontal bones ; situation of wound marked by small puckered cicatrix, in centre of which is an opening admitting No. 1 lachrymal probe ; the probe can be passed for $1\frac{1}{2}$ inches backwards, but in no other direction. There is some epiphora.

CASE 3.—Elizabeth S—, æt. 19, admitted March 10th, 1879. For the last eight months has noticed a lump at the inner angle of the left orbit, and watering of the eye. The lump gradually increased in size, but has always been larger when walking about than when lying down. About four months ago an abscess formed and was opened, the wound never closed, and a constant thick yellowish discharge issued from it. Patient knows of no cause for the swelling ; does not remember having received any injury in its neighbourhood.

On admission.—There is a large dusky swelling at the inner angle of the left orbit ; a constant discharge of thick yellowish semi-purulent looking fluid takes place from an opening in its centre ; a probe introduced through the opening passes downwards towards the lachrymal sac ; there is expansion and apparently thickening of the bones about the inner angle of the orbit ; no displacement of the eyeball. The swelling, though

not in the position of the lachrymal sac was thought to communicate with it.

The fistula was laid open, the upper and lower canaliculi slit, a probe passed down the nasal duct; there appeared to be no stricture. Three weeks later the fistula was closed, and the swelling had greatly diminished.

May 19th, 1879.—Fistula has reopened; swelling as large as ever. Patient readmitted. A free incision made into swelling opened a large cavity, evidently the distended frontal sinus. Some of the bone was chipped away, and a quantity of thick, tenacious, muco-purulent fluid allowed to escape. A probe introduced into the cavity passed across the middle line to the other side of the forehead. The cavity was thoroughly cleared out, a strong iron probe pushed through its floor into the nose, a drainage tube passed through opening thus made, and left with one end protruding from the left nostril, the other from the incision. Some small polypi were found in the left nostril, which might possibly have caused obstruction, leading to distension of the sinus; they had up till now escaped observation. Cavity to be syringed daily with carbolic lotion.

September 8th, 1879.—Drainage tube removed. A quantity of thick mucus is still escaping; swelling has diminished; air passes freely through the opening on expiration with the nostrils closed.

October 6th, 1879.—The swelling has greatly diminished. A cicatrix and small fistula mark the spot where the opening was made; probe passes about an inch through fistula; its end can only be moved about to a limited extent, showing that the cavity is much contracted.

January 12th, 1880.—The fistula has entirely closed; nothing now remains but a little drawing back of the inner canthus. Some thickening of the bones and slight epiphora.

The disease under consideration cannot be so very uncommon, for I have myself treated three cases, and have seen four others the subjects of which declined operative interference; in one of the latter the tumour was very large indeed, and had existed many years.

Yet in many of the text-books, no mention is made of "distension of the frontal sinus" or anything equivalent to it. Thus, in the surgical works of Holmes, Bryant, and Erichsen,

and the ophthalmic writings of Carter and Stellwag—so far as their indices are concerned—the subject is not touched upon.

I suspect that this omission may be partly due to the fact that such cases come indiscriminately under the care of both the general surgeon and the specialist; and in their writings the former have left the description to the latter, and *vice versâ*.

In 'Holmes's System of Surgery,' the disease (if it be the same) is dismissed as follows:—"I would, however, just mention a very singular tumour spoken of by MM. Bérard and Denonvilliers, formed apparently by distension of the frontal sinus, producing intense pain, displacement of the eye, and a large accumulation of gas in the superficial parts of the face, communicating with the neck."

In the following works the disease is treated of under the various names of Distension of the Frontal Sinus, Encysted Tumour, Chronic Abscess or Mucocoele, Enlargement of Frontal Sinus, and Hydatid.

Mackenzie, 'On Diseases of the Eye,' under "Encysted Tumours or Hydatids of the Frontal Sinus," says:—"Professor Langenbeck has published two cases of pressure on the orbit from disease in the frontal sinus. He speaks of them as cases of hydatid, a term much misplaced by German pathologists. Ringer would probably have regarded them as cystic or encysted tumours. Perhaps the one was nothing more than a collection of mucus and the other of thick matter. The situations of the protrusion of the outer table of the bone are amongst the most remarkable circumstances of these cases."

The cases are briefly as follows:

CASE 1.—A female, æt. 17, when eight years of age, in 1802, fell and struck right temple against sharp corner of table. Soon after a hard swelling appeared in region of right frontal sinus. Swelling painless; extended gradually till it involved whole of right side of frontal bone. The right eye became displaced downwards and outwards, vision gradually decreased.

In 1818¹ the swelling was opened. Through the opening there was discharge of clear, ropy, lymphatic fluid, escaping

¹ There appears to be some mistake about the date or age of the patient at the time of operation. She is said to have been eight years of age in 1802, but presumably the report was taken about the same time that the operation was performed; if such were the case she would have been twenty-four, not seventeen.

from a white shining cyst, which filled the whole frontal sinus, and had been penetrated by the perforator.

The cyst or hydatid, as the narrator of the case styles it, was laid hold of with forceps and partially extracted. Measurement of the cavity showed it to be three inches across, and three and a half inches from before backwards. The sinus was filled with lint; injections of willow bark and myrrh, and subsequently of corrosive sublimate, were used.

When the patient left the hospital the swelling had subsided but little. The following year she returned, with the swelling in much the same condition, the discharge of matter being abundant. Two setons were passed through the sinus, by which means the discharge and swelling diminished.

CASE 2.—Male, æt. 20. Eleven years before admission received a stroke with a racquet on the left side of nose and left eye, the consequence of which was a great degree of swelling, which after a time completely subsided.

Two years later he began to have pain, and noticed some protuberance at the inner angle of the eye.

When the patient came to the hospital, vision was unaffected; the eyeball was pressed outwards and downwards by a considerable swelling at the inner angle of the orbit. The swelling had exactly the appearance and situation of a greatly distended lachrymal sac, but was considerably bigger, could not be emptied, nor could any fluid be made to escape from the tear puncta on pressure.

Tumour was cut down on; a white glistening sac came into view. On opening the sac a greyish-white, tenacious fluid escaped; depth of cavity was three inches; finger introduced into it reached as far as the floor of nostril. Termination of the case is not given.

Walton, 'Practical Treatise on Diseases of the Eye,' says, under "Disease of Frontal Sinus:"—"Encysted tumours may be a real dropsy of the cavity or merely a collection of pus or hydatids."

He gives a case of distension of the right frontal sinus by mucus, in a girl æt. 20; the bony wall of the sinus had become absorbed. The swelling was punctured and a small drainage tube introduced; the cavity was frequently syringed out; a fistulous opening remained for a long time.

Gant, 'Science and Practice of Surgery.' "Chronic abscess or mucocele may result when the communication between the ethmoidal cells is closed up and muco-purulent matter accumulates in the sinus." "The swelling may be mistaken for a solid tumour or growth within the sinus, but at a later stage the wall of the sinus becomes thinned and points, and the fluid character of its contents can be felt with the finger."

He recommends that the communication with the ethmoidal cells and nose should be re-established, care being taken to maintain the communication for some days whilst dilute astringent injections are used; that the cavity should be closed as soon as possible, lest a fistulous opening remain, forming with the nasal passage an aerial fistula which would be difficult to close. "A cyst, hydatid or fatty, is sometimes produced in the frontal sinus, giving rise to similar symptoms, and requiring the same treatment."

Soelberg Wells, 'Treatise on Diseases of the Eye.' "Diseases of the frontal sinus may produce considerable dilatation of this cavity, which then encroaches on the orbit, giving rise to contraction and malformation of the latter, and consequent protrusion of the eyeball." Diseases mentioned are—acute and chronic inflammation of the lining membrane, giving rise to purulent or muco-purulent discharge. Polypi, cystic tumours, entozoa, and exostoses are also mentioned. A blow is given as the cause. The treatment recommended: a free incision, thorough evacuation of the contents, a seton passed through between sinus and nasal cavity, and left in for several weeks.

Hulke, 'Royal London Ophthalmic Hospital Reports,' vol. iii, pp. 152, 153, gives two cases of distension of the frontal sinus. One is apparently the same as that reported by Walton. No cause is given. In the other there was a history of injury twelve years before. It was treated by incision; the discharge became purulent; purulent character and quantity of discharge diminished, and at the end of the following month (about six weeks from the time of operation) only a few drops of mucus escaped morning and evening from a small fistula, which the wound had then become.

Two years later the eye was still slightly in advance of its fellow, the orifice of the sinus had become almost capillary, and only occasionally discharged a few drops of clear mucus; some

enlargement of upper part of nasal process of superior maxilla and internal angular process of frontal bone remained.

Lawson, 'Diseases and Injuries of the Eye,' describes "Distension of the frontal sinus" very fully. He gives injury at some time—perhaps very remote—as the most common cause. Two cases are reported: one in a man, æt. 58, in whom the disease was traceable to a kick from a horse fifty-four years before; the other in a woman, æt. 21, in whom the swelling had been first noticed six years before consulting Mr. Lawson, and was attributed to an attack of erysipelas fifteen years before.

The treatment in both cases was by introduction of a drainage tube, and syringing with astringent and disinfectant solutions. Mr. Lawson says the drainage tube should be worn for five or six months, or until all discharge from the nose has ceased.

Bader, 'The Human Eye, its Natural and Morbid Changes,' says: "Most commonly the sinuses are enlarged by accumulation of thick transparent or partly opaque mucus, or by muco-pus, rarely by pus; this may be fetid and mixed with blood." "In rare instances solid bony tumours, exostoses, and polypi attached to the walls of the sinus, or encroaching from neighbouring centres, have been found." Injury was found to be the cause in eight out of nine cases.

The treatment recommended is incision and introduction of a seton through the sinus into the nose. The seton may be removed four weeks after introduction, but in some cases has been left in for several months. In one case the seton, a wire one, set up so much irritation that it had to be withdrawn. The patient daily passed the handle of a cataract knife through the nose into the opening, and "finally succeeded in restoring the normal dimensions of the sinus, and its communication with the nose."

Bader alludes to a case in which some insect had become lodged in one of the sinuses, and caused irritation of the mucous membrane. Benefit was derived from smoking cigars impregnated with arsenic.

Distension of the frontal sinus appears to be caused, in many instances, by a blow about the inner angle of the orbit, causing fracture of bone and subsequent closure of the communication between the sinus and middle meatus of the nose. The blow may have been received at a period very remote from the first appearance of the tumour. In the first of my cases the injury

was more than seventeen years before, and in one of Mr. Lawson's, when the patient was first seen by him, fifty-four years had elapsed since the injury; the report, however, does not say when the tumour was first noticed; it was very large and had probably existed some years.

The great length of time between the cause and its visible effect may be accounted for by supposing that the distension gives rise to no very marked symptoms until the orbital wall of the sinus begins to bulge, or, indeed, until the bone having become absorbed the contents of the sinus point beneath the integuments. The secretion of the sinus is probably only sufficient to keep the surface of its cavity moist, and—providing no inflammatory action was set up—would take years before its quantity was sufficient to cause distension. Moreover, before bulging the external walls the secretion might make room for itself by destroying the partitions between the various cells, not only frontal, but ethmoidal, or by passing across the middle line, and in part discharging itself into the opposite nasal cavities. Thus, in my third case there was evidently a communication between the two sinuses, for a probe could be passed from the incision quite over to the other side of the forehead. In this case, as in Case 2, there was no history of injury, nor could any cause be assigned by the patient for the appearance of the tumour. It is possible that the polypi found in the nose may have blocked the opening of the sinus; they were, however, so small that they had given rise to none of the ordinary symptoms of nasal polypus. If they were the cause of the obstruction they must have grown quite close to, or in, the opening of the infundibulum itself.

Distension of the frontal sinus has probably no early symptoms. There is at no time severe pain, nor indeed any, until the disease has far advanced. In my first case the patient had no pain until after the lump appeared at the inner angle of the orbit, though in all probability the sinus had been gradually filling for sixteen or seventeen years. The pain was described as stretching and bearing down, and was constant. In the other two cases no pain was complained of.

The first symptoms noticed by the patient are swelling about the inner angle of the orbit and perhaps epiphora, the latter being dependent on the former.

A tumour having formed, its nature is not so very evident. In the first of my cases I formed no opinion beyond that there was a collection of fluid pointing above the inner canthus. I did not think it was a distended sac; an incision, followed by the introduction of a probe, showed plainly what it was.

In the second case I diagnosed an exostosis of the orbit, and in the third an abscess connected with the lachrymal sac.

The diagnosis between bony tumour and distension of the frontal sinus—before perforation of the bone has taken place—is not easy. We have, as in Case 2, an irregular tumour projecting from the inner margin of the orbit, hard, and apparently connected with the bone. The tumour feels like bone, grows slowly and painlessly, as bony tumours do, so that both in their physical characters and history the two agree. Later on, however, in distension of the frontal sinus, the bone, already thin enough, becomes thinner, and on pressure upon the tumour a crackling sensation is communicated to the fingers. No such thinning takes place in the exostoses found about the orbit; they are very hard and dense, and feel so.

A correct diagnosis is easily arrived at by cutting into the tumour.

When the bone has become absorbed and the contents of the sinus point, a rounded, fluctuating swelling is formed, which may be mistaken for a distended lachrymal sac. There are, however, certain marked differences between the two. Thus, the position of the swelling in distension of the frontal sinus is different; it is high up at the inner angle of the orbit, above the tendo oculi, instead of beneath it; it cannot, like the distended sac, be emptied by pressure, nor can any of its contents be squeezed out through the canaliculi. At this late stage the tumour caused by distension of the frontal sinus has one marked peculiarity, *it varies in size at different times of the day*. Patients tell us that swelling is much less when they get up in the morning than at other times. This is probably due to the fluid becoming—evenly diffused throughout the sinus, whilst it gravitates to the lowest part after the erect position has been maintained for a few hours. In this stage, as in the earlier ones, an incision into the tumour will clear up any doubt.

Displacement of the eyeball, diplopia, and impairment of

vision may occur or not, according to the size the tumour has reached.

Distension of the frontal sinus is, I think, best treated by drainage. My first case did well with simply an incision through the integuments, clearing out of the sinus, and a counter-opening into the nose. The patient, however, disappeared before sufficient time had elapsed to allow of refilling of the cavity.

The other two cases have been seen occasionally up to the present month (January, 1880). In one the operation was performed ten months, in the other eight months, ago; the drainage tube was left in rather less than three months in Case 2, rather less than four months in Case 3. The result in both is in every way satisfactory, but more especially in Case 2. In Case 3, a small fistulous opening still remains, and there is some thickening of the bones about the inner angle of the orbit.

Since my paper was sent in the following case has been operated on:

Z. P—, æt. 36, first came to me in April, 1876. Had always enjoyed good health; never had any venereal disease. For eight years he had been at sea; for five years subsequently he was a "seaman's labourer;" during the next five years he was employed as a clerk, working long hours in a dark office. When about fourteen years of age patient had a kick above one of his eyes, but he does not remember which. Seven years ago had a blow above one orbit (is almost sure it was the left) from an iron rod connected with a steam hammer. Always enjoyed good sight until he became a clerk (five years ago). Soon after commencing his duties he first began to notice a dull pain at the inner angle of the left orbit, extending up the forehead in the course of the supra-orbital nerve, and along the inner side of the nose; this pain was always worse after he had been engaged for some hours in writing. One day, while in great pain, he covered up his right eye, and then found that the sight was defective in the left. He had some drooping of the left upper eyelid at this time, but is not sure when it first began. His wife had noticed a difference in his eyes for a year or more previously.

When first seen he complained of pain in the left eyeball and orbit, which had been almost constant during the last twelve months; the right eye had become rather painful in the last fortnight. He had ptosis; the note does not say on which side, it was probably the left.

The right eye could read Snellen 40 at twenty feet, and Snellen 30 at the same distance, by the aid of a convex glass of twenty-four inches focus. The left eye could read Snellen 50 at twenty feet without aid, with a convex lens of forty inches focus Snellen 30 at twenty feet. The ophthalmoscope showed hypermetropia in both eyes. He was ordered to use convex 24 for all near work.

He did not get much benefit from the spectacles; the pain in the left eye went on increasing; he went on with his work though advised not to do so; he took iodide of potassium for some time.

On June 28th, 1876, it was noted that there was a good deal of pain about the pulley of the left superior oblique muscle.

On July 19th the refraction was tested more carefully, and astigmatism found in the left eye. Suitable glasses were ordered. He managed to see very well with these, but the pain in the left eye and orbit continued.

No note was made of any swelling about the orbit or displacement of the eyeball. Beyond the ptosis noticed at his first visit nothing but the hypermetropia and astigmatism was made out, and to these the pain and discomfort were attributed.

December 29th, 1879.—During last five months left eye has become prominent. There is now protrusion of the eyeball, which is also pressed downwards and outwards by a painful, semi-elastic swelling, projecting from the inner and upper part of the orbit. A month ago the swelling was inflamed, very painful, and much larger than at present. Vision is somewhat impaired ($\frac{1}{8}$ instead of $\frac{2}{8}$ with spherico-cylindrical lens). There is no diplopia. The ophthalmoscope shows that the inner edge of the optic disc is veiled, and its inner half very red.

Periosteal node diagnosed; ordered Pot. Iodidi gr. xx, Tr. Cinch. Co. mxx, Aqua ʒj, ter die.

February 5th, 1880.—Swelling more solid, decreased in size; no pain except on exposure to cold or attempting to read. Obscure fluctuation in parts. Omit iodide. The swelling is larger at some times than at others, and when patient lies down partially disappears. It was now diagnosed to be the distended frontal sinus.

12th.—Patient placed under influence of an anæsthetic. A hard, immovable tumour could be felt projecting into the orbit from its inner angle; no fluctuation could be detected. The swelling was cut down upon, and a mass of hard bone exposed, in the centre of which was a small opening, from which some thick pus escaped. Some of the bone having been broken away the finger entered a large cavity, and could be passed downwards, backwards, and inwards for about two inches and a half in each direction. A large quantity of thick, greenish, fetid discharge escaped.

A perforation was made from the cavity into the nose, and a drainage tube introduced; some sharp ridges of bone were removed with bone forceps; after this the eyeball returned to nearly its natural position. Cavity to be syringed out daily with carbolic lotion (1 to 40).

16th.—No bad symptoms; some swelling of the eyelids, subsiding. Eyeball pressed slightly outwards and downwards.

23rd.—Noticed impairment of sensation of region of distribution of supra-trochlear nerve (no doubt divided at operation).

March 25th.—No return of sensation; small abscess at upper opening for drainage tube. Optic disc still red at inner side, and margin ill defined.

May 6th.—Has had some inflammation of the upper eyelid and around entrance of tube. There is some thickening of the bone close to the inner angle of the orbit; still some discharge from the lower end of tube. Eyeball in nearly normal position; vision as good as ever. Optic disc rather red, but its outline quite well defined; patient has been at his work as a clerk for some weeks, and by the aid of glasses can write or read for any time without pain or inconvenience. New drainage tube introduced.

In this case there is a distinct history of injury. Some of the symptoms complained of were no doubt due to the hypermetropia and astigmatism, and, as commonly happens, were

first noticed when the patient began to use the eyes continuously upon near work (in his case writing).

The symptoms caused by the anomaly of refraction and those due to stretching of the wall of the frontal sinus were mixed up together; the pain was not entirely due to the former, as it was not relieved by suitable glasses, although the sight was greatly improved. This circumstance, coupled with the existence of ptosis, raised a suspicion that there might be some periostitis about the orbit, or some disease within the skull, but the true nature of the case was never suspected during the whole time that the patient was under observation in 1876.

In 1879 the displacement of the eyeball was evident enough; its cause was also plain, but not so the nature of the tumour.

Unlike the other cases, pain was a very prominent symptom here, and there was one rather sharp attack of inflammation.

Soon after this the patient noticed that the tumour varied in size at different times; this is one of the chief diagnostic signs of "distension of the frontal sinus," and cannot occur until the bone being absorbed the contents of the sinus point beneath the integument.

NOTE
ON THE
TRIANGULAR LIGAMENT OF THE
URETHRA.

BY R. E. CARRINGTON, M.D.

THE connections of the triangular ligament of the urethra are, in my experience, involved in a considerable amount of confusion. That such is the case is constantly being forced upon one's notice in the dissecting room, and consequently I have directed some little attention to this point of anatomy. I have therefore thought it might be of some little use to write this note with the view of endeavouring, so far as I am able, to make the connections of this structure clearer. The difficulty arises, I believe, from two causes: *firstly*, that *identical* parts of the pelvic fascia have received a different nomenclature; and *secondly*, that incorrect, or at all events inexplicit accounts, are given in most of the text-books in ordinary use.

To obviate the first difficulty, I shall take that account of the pelvic fascia which is now commonly received, and indeed followed by the authors usually read; but the connections of the various parts of which with the structure under consideration are either not stated exactly, or are else erroneously given. I mean that description which says that the pelvic fascia divides, at some point in a line extending from the symphysis of the pubes to the spine of the ischium, into two parts, one of which extends in to the various pelvic viscera, and

the other of which is continued down on the inner surface of the obturator internus muscle to the outlet of the pelvis. The line of bifurcation is called the *white line*, the part of the fascia above this line is named *pelvic*, the portion below *obturator*, and the piece extending in to the viscera *recto-vesical*; further, there is a thin fascia derived from the obturator, which lines the under surface of the levator ani muscle, to which the name *anal* is applied.

But whilst laying down these hard-and-fast lines, I will quote here a passage from 'Quain's Anatomy' (vol. i, p. 327), because, whilst I have been repeatedly convinced of its accuracy, I do not think any other of the authors, ordinarily read, mention the facts stated therein. It is as follows:

"The obturator fascia is sometimes included in the description of the pelvic fascia, while the recto-vesical is considered as an offset from it. It will be found however on dissection, that the recto-vesical fascia is always most directly continuous with the pelvic fascia, and that the obturator fascia is only loosely connected with it. Indeed, the fibres of the levator ani muscle in most cases pass upwards to some extent beyond the white line, and thus separate the obturator from the pelvic fascia."

I will now take the various descriptions given of the anatomy of the triangular ligament.¹ It will be found that there is no difficulty about the anterior or superficial lamella, for this is described by all as a *special layer of fascia closing the upper part of the pubic arch*. It is in the connections of the deep layer that the discordant accounts, real or apparent, are given.

Thus, in "Gray's Anatomy" (p. 751) it is stated "the posterior layer is *derived from the pelvic fascia*," and again (pp. 755-56) the *pelvic fascia* "is continuous below the pubes with the fascia of the opposite side, so as to close the front part of the outlet of the pelvis, blending with the posterior layer of the triangular ligament:" and further, the obturator fascia "is a direct continuation of the pelvic fascia below the white line, and is *attached to the pubic arch*." The inference from this account is, I take it, that the posterior layer is formed by the *pelvic fascia* closing the pelvis in front and beneath the symphysis, and that the obturator division takes no part in the

¹ The italics are my own in all the succeeding quotations.

formation of the triangular ligament, but stops short at the pubic arch.

In 'Quain's Anatomy' (vol. i, p. 326) we find "this layer (i.e. deep layer of triangular ligament) is superficial to the anterior fibres of the levator ani, which lie between it and the *pelvic fascia*,¹ and is connected with a thin web of areolar tissue, which extends backwards on the surface of the levator ani muscle and is distinguished as the *anal fascia*." The obturator division is described as being attached to the rami of the pubes and ischium.

There is nothing said as to the connection of the triangular ligament with the pelvic fascia, or with any part of it, and I think that we must either infer that both layers of the ligament are to be looked upon as *special fasciæ*, or that else the deep layer is derived from the *anal fascia*.

In 'Holden's Manual' (p. 369) the matter is very briefly disposed of, and it is simply stated that "the posterior layer (of the triangular ligament) is a part of the *pelvic fascia*."

In Heath's account of the triangular ligament, no mention is made of its connection with any part of the pelvic fascia, but in describing the *recto-vesical layer* (p. 261), the following is stated: "It is seen to dip down to the prostate, and is thus continued from one side to the other of the pelvis, of which it closes the outlet."

In 'Ellis's Anatomy' (eighth edition, p. 546), no distinction is made between pelvic and obturator fasciæ, but the *whole membrane lining the inner surface of the obturator muscle is called pelvic*, and the following statement is made:—"Inferiorly the fascia is attached to the hip-bone along the side of the pubic arch." He is here speaking of the part *below the white line*, i.e. the obturator division of other writers. On p. 549, it is stated that the recto-vesical fascia between the pubo-prostatic ligaments of opposite sides "*dips down to reach the triangular ligament of the perinæum, and closes the pelvis between the levatores ani*." On p. 429 we find, "the posterior layer (of the triangular ligament) is derived from the *recto-vesical fascia*. Here, then, we have another account of the origin of this layer.

¹ The word *pelvic* must here be read in connection with the passage quoted above (vol. i, p. 327). *Recto-vesical* is probably meant as being a direct continuation of the *pelvic fascia*.

I may here note that in the sixth edition of this writer's work (p. 587) the account given of the pelvic fascia differs substantially from the later one just quoted; in that it (the pelvic fascia) is stated to be "*continued from the one hip-bone to the other, so as to close the cavity of the pelvis in front for a short distance;*" and that on p. 458, it is said, that "*the posterior layer (i.e. of the triangular ligament) is derived from the pelvic fascia.*" We must however of course take the later description, which gives the origin from the recto-vesical fascia.

I think I have quoted enough to justify the statement that the connections of the posterior layer of the triangular ligament may well be a stumbling-block, for we have the following different accounts given:—

1. From the pelvic fascia (Gray and Holden).
2. A special structure, or from the anal fascia (Quain).
3. From the recto-vesical fascia (Ellis).

It seems to me that the following considerations may make the matter clearer:

1. The pubic arch is of course below the level of the symphysis.
2. The white line extends from the *lower part* of the symphysis pubis.

Therefore the *triangular ligament must be below the level of the pelvic fascia*, which only extends as far down as the white line.

I believe the true account, and it is one which I have verified by dissection, to be as follows:—

The superficial layer is a *special fascia*. All accounts here agree. The deep layer is formed by the obturator fascia, which anteriorly is found to stretch across the pubic arch and close it; or perhaps a little more exactly, the obturator fascia may be described as running down to be fixed to the margin of the pubic arch, and a little above its attachment, to give off a process which passes across the upper part of the arch, and which, joining with a like process from the fascia of the opposite side, forms the deep layer of the triangular ligament. An additional proof that this is the true formation of this layer is found in the fact that the triangular ligament extends as far back on each side as the tuber ischii, where it is, of course, far below the level either of the pelvic, recto-vesical, or anal fasciæ.

FIG. 1.

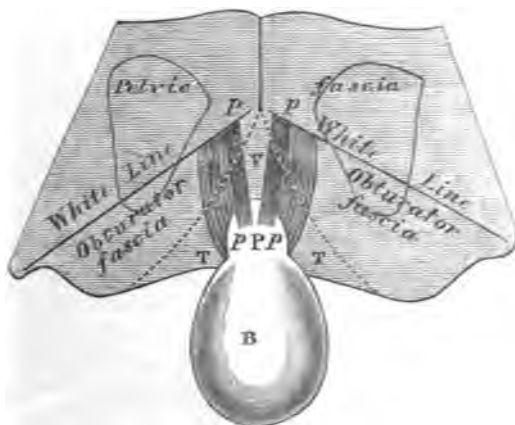


DIAGRAM OF THE POSTERIOR SURFACE OF THE SYMPHYSIS PUBIS, SHOWING THE DIVISIONS OF THE PELVIC FASCIA, AND THE CONNECTIONS OF THE POSTERIOR LAYER OF THE TRIANGULAR LIGAMENT.

B. Bladder drawn backwards.

P. Prostate.

p, p. Pubo-prostatic ligaments.

L, L. Anterior fibres of the levatores ani muscles, arising from the lower part of the symphysis pubis.

T, T. The posterior layer of the triangular ligament, continuous laterally with the obturator fasciae.

The white lines correspond to the cut edges of the recto-vesical fasciae, and the pubo-prostatic ligaments to the anterior edges of the same. Between these two there is a triangular interval *v* in the median line, in which the recto-vesical fasciae dip down to join the posterior layer of the triangular ligament and the capsule of the prostate.

The pubo-prostatic ligaments are on a higher level than the levatores ani muscles.

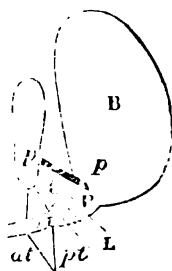
If, therefore, in Gray's account, for *pelvic* we write *obturator*, it would be correct. The triangular ligament *is* continuous behind with the anal fascia, but I do not think that the second alternative of Quain's description is right, viz. that the posterior layer is derived from the anal fascia, for this would be tracing a comparatively thick membrane from a very thin one; and as to the first, that it is a special structure not connected with any division of the pelvic fascia, I would say that I have often traced its continuity with the obturator, as follows:—

If the obturator fascia be separated from the outer wall of the ischio-rectal fossa, and the dissection is carried forwards

along the pubic arch, keeping close to the bone, the triangular ligament will also be detached; the knife will not pass between these two structures, separating them from one another, but they will be seen to be directly continuous, and to be dissected off in one piece.

Finally, we have Ellis's derivation from the recto-vesical layer. This to my mind cannot be the case, for, as is indeed expressly stated by Quain, the fibres of origin of the levator ani muscle from the symphysis pubis are found distinctly between the recto-vesical fascia and the posterior layer of the triangular ligament.

FIG. 2.



ANTERO-POSTERIOR VERTICAL SECTION THROUGH THE BLADDER AND SYMPHYSIS PUBIS.

B. Bladder.

P. Prostate.

M. Membranous urethra.

p. p. Pubo-prostatic ligaments.

L, L. Levator ani, arising from the symphysis and placed between the pubo-prostatic ligament (i.e. recto-vesical fascia, and the posterior layer of the triangular ligament).

It is however a fact, that the recto-vesical fascia closes the pelvis *above* these attachments of the levator ani, between the anterior portions of the two white lines, except in the centre, where it dips down to join the prostate and the deep layer of the triangular ligament, leaving a small interval; but this makes another layer closing the pelvis just below the symphysis, and we have, indeed, from without inwards :

1. The superficial layer of the triangular ligament.
2. The deep layer of the same, derived from the obturator fasciæ.
3. The recto-vesical fascia.

And between the recto-vesical fascia and the triangular ligament a median septum is formed by the former dipping down in the centre to join the latter. On either side of this there is a little space, bounded above by the recto-vesical fascia, below by the deep layer of the triangular ligament, and internally by the median septum, externally by the obturator fascia covering the pubic bone, and in this the anterior fibres of the levator ani muscle run up to the symphysis.

This may readily be demonstrated by carrying the ordinary dissection of the perinæum a little further. If the posterior layer of the triangular ligament, which is usually the deepest limit taken, be removed, this little space is opened up, and, from without inwards, first the levator ani muscle is met with, and deeper still the recto-vesical fascia.

Conversely, if a dissection be made similar to that indicated in the diagram, Fig. 1; when the recto-vesical fascia is removed the anterior fibres of the levator ani muscle at their attachment to the symphysis are exposed, and if these be then taken away, the posterior layer of the triangular ligament is uncovered.

The above description is, in its essential details, just as it was written for another purpose some months ago. Since that time I have consulted Henle's '*Handbuch der Anatomie*,' vol. ii, part 1. The account of the triangular ligament, as given by this author, is different to that of English writers, for he describes the two layers, with the intervening muscles, as "*the uro-genital diaphragm*," which, on page 406, is stated to be "a partly aponeurotic partly muscular plate, stretched across between the lower margins of both pubo-ischial bones."

Again, on page 408, the muscular portion is stated to be the deep transverse muscle, and the two layers of the triangular ligament, one of which, of course, is above and the other below the muscle, are described as its upper and under fasciæ respectively, and not as a ligament at all.

On page 525 we find that "the upper surface of the deep transverse muscle of the perinæum is covered with a layer of fascia, which bends upwards to the lateral borders in the obturator fascia, and passes towards the middle line on to the prostate, and from this to the sides of the urinary bladder."

Now, Henle calls all the fasciæ covering the obturator

internus muscle *obturator*. Nevertheless, it is, I think, evident from the drawing (fig. 432) that the portion alluded to in the above quotation is the part below what we know as the white line, and that, therefore, thus far his account agrees with mine. But he says also, it passes at the middle line to the prostate and urinary bladder, and again, in another place, "the upper layer (of fascia of deep transverse muscle) is on both sides reflected from the pubes on to the prostate, and serves as a covering for the latter." Now, in my account I have said that the recto-vesical fascia dips *down* in the median line in front to join the prostate and deep layer of the triangular ligament, and I think this is better than stating that the deep layer passes *up* to the prostate and urinary bladder, which is what in effect, I believe, Henle says. A reference to his drawing (fig. 402) will, I think, make this still more evident.

Quoting further, on pages 525 and 526, we find:—"Between the wall of the pelvis and the bladder there is a deep, narrow depression, the floor of which forms the upper fascia of the deep transverse muscle. In this depression the levator ani runs backwards, with its lower border resting upon the upper fascia of the deep transverse muscle, and with the lowest bundles of fibres arising from the foremost part of the same."

Now, I believe this "deep, narrow depression" must be the little space I have described on either side between the recto-vesical fascia above and deep layer of the triangular ligament below, in which the levator ani muscle runs up to the symphysis. Henle calls it "a depression" (literally a valley), but the anterior fibres of the levator ani are certainly separated from the pelvic cavity by the recto-vesical fascia, in addition to the floor being formed by the upper fascia of the deep transverse muscle. He should, therefore, I think, have added was a roof constituted by the recto-vesical layer.

I have quoted Henle thus fully, because I believe that his account substantially agrees with what I have ventured to put forward as the true one. Nevertheless, it is difficult to compare him with the English writers with regard to this structure from want of uniformity in nomenclature and treatment. I am not sure, indeed, whether it may not be considered that my interpretation of his words is rather strained. One naturally wishes to have the confirmation of so distinguished an

anatomist; and, certainly, if his text does not bear the construction I have put upon it, it is distinctly at variance with the plate he gives to illustrate the arrangement of the structure I have been considering. The references, however, are pages 525 and 526. The drawing, fig. 402, page 525, and pages 436 *et seq.*

Finally, I find, also, that Godlee, in the text to part vi of his plates, makes the following statements (p. 220):—"It (obturator fascia) is to some extent continuous along the arch of the pubes with the posterior layer of the triangular ligament." And, again (p. 221):—"The posterior layer of the triangular ligament * * * * at its attachment to the pubic arch may be said to be continuous with the obturator fascia."

REFLEX ACTION IN DIAGNOSIS.

By P. HOBROCKS, M.D.

TAKING part in an ordinary reflex action are the following :

- a.* Stimulus.
- b.* Peripheral termination of a sensory nerve.
- c.* Sensory or afferent nerve.
- d.* Ganglion centre.
- e.* Motor or efferent nerve, with its peripheral termination.
- f.* Muscle or other irritable tissue.

Particular attention is called to the fact, that the muscle itself is a part of the reflex loop, and therefore, when no action results from a stimulus, which usually produces an action, the lesion may be in any part of the loop, including the muscle.

The visible, palpable muscular action itself, normally produced by stimuli, may be diminished or increased, or there may be no action at all to any stimulus, or there may be no action to certain stimuli, and greater or less action than normal to other stimuli.

Physiologists tell us that a reflex act is influenced by the intensity, number, and duration of the stimuli applied to the sensory nerves ; and, as a rule, the resulting movement is more complex when the stimulus is applied to the peripheral nerve endings than to the nerve trunk. Practically, only the former are stimulated by the physician for the purpose of diagnosis.

The stimuli are under his control. He can apply one or more of them in any quantity and for any length of time. No other part of the reflex process can be influenced in this way, although, as will be mentioned further on, he may modify certain other parts indirectly. If, then, a certain definite stimulus be used, and applied for a given time to a certain part on one side of the body and then on the other, and the resulting actions be compared, any difference observable between the two must depend upon a difference somewhere in the reflex loop involved. Again, when it can be stated that, in a normal

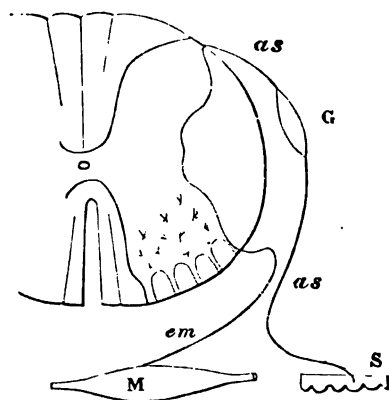


FIG. 1.—SECTION SHOWING LEFT HALF OF SPINAL CORD.

From the anterior cornu is seen coming off an efferent motor nerve (*em*), passing on to a muscle (*M*). From skin (*S*) is seen ascending to the posterior cornu an afferent sensory nerve (*as*), with the ganglion (*G*).

state of health a certain stimulus, applied to a certain part, will produce a certain action, then, if such action do not result in a patient, or if it be diminished, or sluggish, or exaggerated, it may also be asserted that the reflex loop is affected in some way. Nevertheless, it must be remembered that persons differ widely in reflex irritability, that in children it is greater than in adults, probably because in them the brain is not so highly developed, and so the reflex actions in the spinal cord are not so much under control; also, that conditions of the skin and superficial fascia, such as a thick skin, a large quantity of adipose

tissue, œdema, &c., may greatly modify reflex actions without any real disease in the reflex loop, beyond such physical interference with the ends of the sensory nerves as would be caused by the above.

In the annexed diagram (Fig. 1) are seen the various parts entering into the reflex loop. The afferent sensory nerve passes through the posterior root into the posterior grey cornu, or into the lateral column in its immediate neighbourhood, whilst the efferent motor nerve is issuing from the anterior grey cornu and passes through the anterior root, and then onto the muscle. It will be noticed that the motor and sensory nerves run close together to their destinations; and, as a matter of fact, they are generally bound up together so as to form one nerve apparently, as in most of the spinal nerves. But this is not at all essential; they may run quite separately, as in most of the cranial nerves, and even in the spinal nerves, where they run together, they are perfectly distinct, never anastomosing together. Theoretically the reflex loop may be affected at any part, and hence we might have lesions of:

1. Peripheral endings of sensory nerves.
2. Sensory nerves (including posterior roots).
3. Grey matter of cord

{	Anterior cornua.
	Posterior cornua.
	Portion between these two.
4. Motor nerves (including anterior roots).
5. Peripheral endings of motor nerves.
6. Muscles.

And again, the grey matter of the cord may be influenced by disease in any part of the white matter of the cord; that is, the posterior, lateral, or anterior.

It must also be remembered that a ganglion centre which is receiving sensory impressions, is not so readily traversed by another sensory impression arriving from another part. A person, for instance, under great mental excitement or pain might have his ordinary reflexes abolished; and thus may be explained, what has been noticed a great many times, that soldiers in battle may receive blows and wounds of which they are entirely ignorant until after the battle, the stimuli having neither reached the higher perceptive faculties, nor produced the ordinary reflex results.

Experimentally, in animals, any one of the six parts of the reflex loop above enumerated can be irritated or destroyed ; and practically, one meets with instances of lesions of each, except, perhaps, the peripheral terminations of motor nerves ; and, perhaps, as pathology advances, such lesions may be found. Thus we get lesions of the peripheral ending of the optic nerve in retinal disease ; of sensory nerves, as in tumours of the different trunks of the fifth ; of the grey matter of the cord, as in anterior and posterior cornual myelitis ; of motor nerves, as in facial paralysis ; of muscles, as in inflammation of muscle, flabbiness due to generally weak conditions of the system, &c.

Experimentally the motorial end plates can be paralysed by curare.

1. Skin and mucous membranes.

The ordinary stimuli applied to these parts give rise by reflex action to certain movements. Thus, tickling the soles of the feet causes drawing up of the lower extremities. A scratch on the inner side of the thigh produces contraction of the cremaster muscle. A scratch from the last rib to the anterior superior spine of the ilium causes contraction near the umbilicus, and one from the nipple to the hypochondrium causes a movement in the epigastrium. Similarly scratches in various other regions cause contractions in those parts severally. In the mucous membranes the reflexes are much more complex. Thus, tickling the back of the fauces causes vomiting, tickling any part of the larynx causes coughing, tickling the nasal mucous membrane causes sneezing. These reflexes have been described as superficial reflexes in contradistinction to the so-called deep reflexes (Gowers), but inasmuch as the latter are probably not reflex, the term "cutaneous reflex" is preferable, or if "superficial reflex" be retained, it must be taken as meaning that the stimuli are applied to superficial portions.

From the above the following names will be easily comprehended :

Plantar reflex.

Cremaster ,,

Abdominal reflex.

Epigastric	„	
Gluteal	„	
Lumbar	„	
Dorsal	„	
Scapular	„	
Palmar	„	
Conjunctival	„	
Pharyngeal	„	(retching).
Laryngeal	„	(coughing).
Nasal	„	(sneezing).

In obtaining these cutaneous reflexes, it is best to pass the tip of the finger, or the point of a quill pen, rapidly and lightly over a rather large surface, so as to stimulate quickly and nearly simultaneously a great many endings of a nerve. In children it is better to use the finger than a pen, especially in getting the cremaster reflex, for if present, it is most easily obtained in this way, and they cry if scratched with a pen.

The first four in the above list, and the conjunctival reflex, are the most useful in diagnosis. For they are, as a rule, present in normal individuals and are more easily observed than the rest.

Mention has been made of the fact that the centres in the spinal cord are in communication with the higher cerebral and cerebellar centres. When these are cut off, as by section through the spinal cord, the cutaneous reflexes below the section are increased. Thus, a man fractures his spine and injures the spinal cord, say in the dorsal region; tickling the soles of his feet produces much more violent movements of the lower extremities than under normal conditions; and it is on this account that the brain is said to inhibit the reflex action of the spinal cord. In animals, when the cord is cut, the reflexes below are abolished for a time, apparently from the shock of the operation; but after a certain period of time, which is very short in frogs, but some weeks in dogs, the reflexes are much more violent than is normal. Similarly in fracture of the spine the plantar reflex may not be increased for some weeks.

The reflexes may be increased in disease, as in tetanus, where the slightest stimulus produces general contractions, the apparent impulse flowing readily into the various motor channels;

and in poisoning by strychnia, where the same reflex irritability is seen.

These cutaneous reflections are not altered in :—

Meningitis.

Paralysis agitans.

Hydrocephalus.

Chorea.

Epilepsy.

Hysteria.

I have tested them in all these diseases, and they have invariably been present, except, perhaps, the cremaster reflex, which has sometimes been absent in adults with a very loose and long scrotum.

It is to be observed that in none of the above diseases, so far as is known of their pathology, is the reflex loop affected, nor the cerebral connection of the cord interfered with. Nevertheless, it is possible that, hereafter, cases of hysteria may be found in which the cutaneous reflexes are altered. I have not yet had an opportunity of testing them in a case of pseudo-hypertrophic paralysis, and have not been able to find any observations on the point; they would probably be absent, at least in the parts affected, seeing that the muscles become practically destroyed.

If cutting off the higher cerebral centres causes an increase in the reflex phenomena of the cord below, we should expect the cutaneous reflexes to be increased, not only in fracture of the spine, but also in

Transverse myelitis.

Pott's disease.

Growths.

Gummata, &c.

No doubt this would be the case if it were not that descending lesions are so rapidly induced, which lesions modify the reflexes.

It is a remarkable fact that the cutaneous reflexes are diminished on the paralysed side in hemiplegia due to cerebral origin, and not so when the paralysis is due to a lesion in the cord. Dr. Gowers suggests the ingenious theory that it is due to a cutting off of a centre in the cortex of the brain which normally inhibits an inhibiting centre in the optic thalamus. The fact may be of use in diagnosis, though all the cases that

have yet come under my own observation were easily diagnosed as cerebral, quite apart from the reflexes.

The cutaneous reflexes are also diminished in

Infantile paralysis (*e.g.* plantar reflex abolished when the leg is affected).

General spinal paralysis.

Progressive muscular atrophy.

Tabes dorsalis.

Local myelitis.

Lesions of motor or sensory nerves.

In the first three there is not only a lesion in the reflex loop (in the anterior horns of the cord), but also a wasting of the muscles which perform the act.

In tabes dorsalis these reflexes are not affected at first but only after the disease has made some progress. The plantar reflex is one of the first to disappear, and there is, as a rule, a considerable amount of anæsthesia. This is probably due to the fact that the posterior roots are involved in the sclerosis which constitutes this disease. But it must be remembered that anæsthesia may be due to a lesion in a part of the spinal cord above, in which case the cutaneous reflexes below would be increased; in other words, it must not be assumed that where there is loss of sensation there is also an absence of the cutaneous reflexes.

The cremaster reflex is the next to disappear, but for a long time, and it may be to the end, none of the rest disappear, probably because the sclerosis is not wide enough, and so the reflex loop is not affected, except in the lumbar part of the cord.

In local myelitis the cutaneous reflex, having its centre in the affected part of the cord, is abolished. Those below it are increased, and those above it remain normal.

In lesions of sensory nerves there is anæsthesia as well but no paralysis, whilst in lesions of motor nerves there is paralysis but no anæsthesia. Lesions of mixed nerves produce both paralysis and anæsthesia.

As an example of absence of reflex in lesions of sensory nerves may be taken affections of the fifth nerve. Irritation of the conjunctiva no longer produces contraction of the orbicularis palpebrarum; at the same time there is anæsthesia of

the face. Again, the lesion of a motor nerve is illustrated by facial paralysis, in which irritation of the conjunctiva produces no action because the orbicularis palpebrarum is paralysed, along with all the other facial muscles.

It is said that sexual power varies *pari passu* with the cremaster reflex. This is verified in many cases of locomotor ataxy, where complete absence of the cremaster reflex is accompanied by complete absence of sexual appetite. Whilst in certain other cases where it is increased there is an admitted increase in the sexual desire. It is not, however, always the case.

Patients will be met with having almost complete loss of sexual desire and power (the desire generally fails a considerable time before the power), whilst the cremaster reflex is normal; and it is not uncommon to find the latter absent with no change in the sexual functions.

It is a difficult matter to explain why, in certain cases, the so-called deep reflexes are increased and the cutaneous reflexes diminished. But, as will be described further on, the former are in all probability not true reflex phenomena, though depending on the integrity of the reflex loop for their production, whilst the latter are certainly truly reflex, inasmuch as the resulting muscular action may be quite removed from the seat of irritation; thus, tickling the soles of the feet causes contraction of the thigh muscles; stroking the middle of the upper part of the thigh causes contraction of the cremaster. In both of these cases the muscular fibres are not directly irritated, nor are their tendons at all pulled upon. It is possible also that the reflex loop between skin, cord, and muscle, is not the same as that involved in the so-called deep reflexes, which, as will be presently mentioned, is probably from muscle to cord, and thence by motor nerves to muscle again.

2. Muscles, tendons, &c.

If, with one leg crossed over the other the ligamentum patellæ be struck with the side of the extended hand, the foot is kicked out involuntarily. This occurs in almost every one.

A few years ago Westphal, of Berlin, pointed out that this action could not be obtained in locomotor ataxy. Almost at the

same time Erb wrote upon the same subject, and described the normal phenomenon as the "patellar-tendon reflex," believing it to be a reflex act. Westphal called it the "knee phenomenon," and did not think it to be truly reflex, but to result from an irritation of the muscular fibres, due to the pull on the tendon caused by the blow upon it.

Dr. Grainger Stewart and Dr. Buzzard were the first to describe it in this country, and they took Erb's view of the case, namely, that it is a reflex phenomenon.

Westphal then pointed out that if it was reflex the afferent nerves could not be cutaneous (patellar plexus) nerves; for, not the slightest action can be obtained by any amount of pricking or otherwise irritating the skin, or percussing it when pinched up in a fold. The afferent fibres were then said to be in the tendon itself. This, however, has been disproved, at least in the tendo-Achillis, by Dr. Gowers in his work on the 'Diagnosis of Diseases of the Spinal Cord.' It must be mentioned that if the tendo-Achillis be struck, a contraction of the gastrocnemius results, just like that of the quadriceps extensor when the ligamentum patellæ is struck. "While a lateral tap on the tendon (tendo-Achillis) will cause the contraction, if the other edge of the tendon is so supported that the tendon cannot move under the tap, and so cannot affect the muscle, no contraction occurs." This proves that the afferent nerves are not those in the tendon.

Another important fact confirming the same thing is, that no other kind of stimulus, electrical, chemical, or thermal, applied to the tendon, will cause a contraction of the muscle.

The next supposition by the reflex theorists was, that the afferent nerves are in the muscle itself (see dotted line in fig. 2), and that the blow on the tendon, by pulling upon these nerves, originates a stimulus in them which passes up to the spinal cord, along the sensory tract, and therefore through the posterior roots, and thence from the spinal cord along the motor nerve to the muscle.

Tschiriew then pointed out that the time between the blow on the tendon and the resulting contraction was much too short (only .03 of a second) for it to be a reflex act, according to the known rate at which nerve impulses travel. Dr. Gowers also pointed out that the time in the closely allied ankle clonus, which will be described presently, was much too short for that

to be truly reflex, and quite recently Dr. Waller¹ has confirmed Tschiriew as to the shortness of time in the production of the knee phenomena, and has made the important observation that the actual time taken to produce contraction of a muscle is exactly the same (viz. .035 sec.) in

(a). Percussing its tendon.

(b). Percussing its muscular fibres.

These facts seem fatal to the reflex theory, unless it be assumed that the rate of nervous impulse is much quicker than physiologists have yet made out. But besides the reflex theory there is another, which may possibly be the correct one. I will designate this the "tonic theory."

Normally muscles are in a state of slight contraction, which physiologists call tone. And this tone depends for its existence not only upon a healthy condition of the muscles themselves, but also upon a healthy condition of some reflex loop, between either skin, cord, and muscle, or tendon, cord, and muscle, or muscle, cord, and muscle (*vide* Fig. 2).

Whatever be the reflex loop, it is quite certain that, whenever it is cut, the muscle loses its tone. Brondgeest, Cyon, and others, have shown that when the sensory nerves of a part, or the posterior roots (also sensory), are cut, the corresponding muscles lose their tone.

When the spinal cord is destroyed and broken up the muscles lose their tone, as can be easily demonstrated by passing a wire down the spinal canal of a frog and breaking up the cord, and, as is constantly seen in cases of acute myelitis, which destroys the cord, so far as the reflex loop is concerned. Again, when the motor nerve is cut or diseased, the muscles lose their tone; *e.g.* in facial paralysis due to inflammation about the stylo-mastoid foramen, the muscles of the affected side lose tone and therefore drop, the healthy side appearing to be drawn up. Lastly, if the peripheral ends of the motor nerves be paralysed by curare, the muscles become perfectly flaccid and toneless. So that both experiment and disease declare that, whenever and wherever the reflex loop is destroyed, the muscles lose their tone and become flaccid.

One can also make the converse statement "that whenever a muscle has lost its 'tone' the reflex loop is somewhere

¹ 'Brain,' part x, July, 1880, p. 179,

affected," if the muscular fibres which may lose their tone from disease in themselves, or from exhaustion, be understood to form part of the reflex loop.

A blow on the tendon of a muscle will increase the tension in that tendon, and as a consequence pull upon the muscular fibres above ; and if these muscular fibres be in a state of slight tension, *i.e.* tone, before the pull, then they will contract upon the additional tension being applied, but if they be flaccid, the additional tension is not sufficient to irritate them into a contraction.

Dr. Buzzard pointed out, however, that in locomotor ataxy, whilst there was complete absence of contraction in the quadriceps on percussing its tendon, yet on percussing its muscular fibres directly it contracted as well and often more readily than is normal. This I have confirmed in several cases. There is, however, a difference between a direct blow on the belly of a muscle and a pull upon those fibres from the tendon below.

I have searched in works on physiology to see if a pull on the tendon is one of the ways in which a muscle can be made to contract, but can find no allusion to it. So I made the following experiments, assisted by my friend Mr. J. H. Barnard :

Experiment 1.—A frog was pithed, and then one of its muscles removed, together with the piece of bone from which it took its origin ; the bone was then fixed to a peg and the tendon pulled over a small iron bar and a weight hung to the end of it by means of a piece of silk tied round the tendon. Letting fall the weight or pulling upon it in any way failed to produce the least effect, although the muscle was in a perfectly healthy and irritable condition, as was shown by the ready contractions produced by slight blows on the fleshy muscular fibres.

The experiment was varied by suspending the muscle perpendicularly and pulling upon the tendon below. There was not the slightest response. Of course the muscle being removed from the body was quite flaccid and toneless.

Experiment 2.—The same muscle was exposed in the opposite limb, and its tendon of insertion separated so that a silk thread could be passed round it. None of the nerves were interfered with, nor the spinal cord, beyond the pithing above. So that the muscle was in a normal state of tone, and the reflex loops intact. The limb was then placed over a bar so as to hang

perpendicularly, the body of the frog being supported on a table. A pull upon the tendon, or simply hanging a weight upon the thread attached to the tendon, readily caused contraction in the muscle.

The first of these experiments shows clearly that a pull on the muscular fibres from the tendon below will not cause contraction in a muscle, whilst a blow on the fibres themselves will do so. I do not know of any explanation of it, because it is clear that the pull must irritate the muscular fibres, in some degree at least.

Seeing that the reflex loop had to be left intact in the second experiment, it may be said that the pull on the tendon caused a contraction in the muscle, not by irritating the fibres directly, but by irritating afferent nerves, the stimulus passing up to the cord and down the efferent nerves by reflex action. But I would ask the reflex theorists, why the pull on the tendon below will not set up contraction in a muscle removed from the body, or one whose reflex loop is destroyed. The fibres must be pulled upon, and they are irritable to direct percussion. But it is clear that a direct blow is very different from an indirect pull.

That the absence of contraction upon striking the tendon of a muscle is due to loss of tone in that muscle is true, I believe, for the following reasons :

1. It is absent when the afferent nerves to a part are cut. Brondgeest has shown that, under this condition, the muscle has lost its tone.

2. It is absent in locomotor ataxy, where the posterior root zones are affected.

Debove and Boudet have shown that there is always loss of tone in the quadriceps and various other muscles in tabes.

3. It is absent in all cases of anterior cornual myelitis (infantile paralysis, adult spinal paralysis, &c.), and in all of them there is loss of tone.

4. It is diminished or absent when the muscles themselves are partially or completely toneless, either from excessive use, or disease in themselves.

This I have proved in myself after a long walk ; and indeed there is less response at night after a day's work than in the morning.

Since the above was written I have been pleased to see an

observation, made by Westphal, in the 'Berliner Klin. Wochenschrift,' Jan. 3rd, 1881, which is confirmatory of my statement. It is that the knee phenomenon is wholly absent just after an epileptic fit. It is obvious that during the fit the muscles are most powerfully contracted, both during the tonic and clonic state; and hence they become tired out, and relax into tonelessness, and so the tendo-muscular phenomenon is absent.¹ But the muscles soon recover, and hence the normal phenomenon speedily reappears. Dr. Gowers tells me it is absent for thirty seconds only.

It is interesting also to observe that in most of the cases published by Dr. Gowers in which the knee phenomenon could not be obtained, quite apart from any spinal disease, the patients were bad walkers, probably because their muscles were easily exhausted.

5. If an animal be curarised and pithed, all its motor nerves are paralysed at the peripheral extremities, and the muscles lose tone.

I have done this with the assistance of my friend Mr. Barnard, and we found complete absence of the tendo-muscular phenomena.

It is obvious, however, that none of the above reasons disprove the reflex theory, indeed, they might equally be used as arguments in its favour. For if this theory be true it is obvious that a lesion of any part of the loop will cause the disappearance of the phenomenon. And again, in those cases where the muscle itself has become flabby from overwork, walking, epilepsy, &c., the reflex theorists might say, that of course no contraction will result, no matter what stimulus passes round the loop, if the moving portion, i.e. the muscle, be from any cause rendered useless. At the same time, one would have thought that if the stimulus be greatly increased it might be able to whip up a flagging muscle to contraction.

But every tittle of evidence brought thus far in favour of the reflex theory is equally in favour of the tonic theory, and *vice versa*; and there then remain these facts which, to me at least, seem to turn the scale in favour of the latter.

6. The time is much too short for it to be reflex, whilst it is

¹ The fit must be a severe one with great struggling, or else the phenomenon is not absent.

exactly what one would expect, if the contraction of the muscle were due to the direct irritation of the fibres by the pull on the tendon.

7. The actual time taken to produce contraction of a muscle is, as has been already mentioned, exactly the same in :

a. Percussing its tendon.

b. Percussing its muscular fibres (Waller).

But, whichever theory be true, the usefulness of the phenomena in diagnosis is in no wise impaired ; for inasmuch as the absence or exaggeration of the contraction is in nearly all cases due to an alteration in the muscle produced by a lesion in the reflex loop, one can readily diagnose such a lesion to be present.

These tendo-muscular phenomena are found normally in all the muscles in the body whose tendons are, under the necessary conditions, that is, capable of being percussed or pulled or otherwise stimulated, so as to increase their tension. Hence we find the best examples in the quadriceps extensor with its ligamentum patellæ ; the gastrocnemius and soleus with the tendo Achillis ; the triceps with its tendinous insertion into the olecranon. If the outer side of the back of the wrist be percussed, there is sometimes a direct flexion of the forearm on the arm, which is due to contraction of the biceps, set up by the pull on its tendon where it is inserted into the tuberosity of the radius, besides which there may be an extension of the hand from contraction of some of the extensors.

Other muscles are not so readily excited, but the zygomatic muscles and others around the mouth may be made to contract by percussing their tendinous attachments (malar bone, jaw, &c.). Hence we may speak of these phenomena as—

a. Knee phenomenon.

b. Ankle phenomenon.

c. Elbow phenomenon.

d. Wrist phenomenon.

e. Zygomatic, and other phenomena.¹

The knee phenomenon is said to be absent in normal individuals in one per cent. (Berger). Westphal, however, has never yet met with a case, and out of more than 200

¹ Since the above was written Dr. Gowers has proposed the general term "myotatic contraction" (*τατισμός*, stretched), one of the essential conditions being a certain degree of tension in the muscle.

cases in which I have tried it, it was only absent in those who were very fat, and whose ligamentum patellæ could not be properly got at; and in most of those cases no action could be obtained on direct percussion over the belly of the muscle itself, the same condition being present, *i.e.* a thick layer of fat. I may also state that it is exceedingly difficult to obtain the phenomenon in some people, who are wholly unable to relax their muscles sufficiently, for any voluntary contraction at once stops the phenomenon. By far the best plan of obtaining it, and in no case ought the phenomenon to be called absent unless it be adopted, is to have the patient seated on a high table, with boots, stockings, and trousers off, and the legs hanging loosely down. Several times I have got it in this way when both others and myself were unable to obtain it in any other.

The amount of contraction in the muscle, and therefore the height to which the point of the toe rises, varies in different people.

The ankle phenomenon, *i.e.* contraction of the gastrocnemius and soleus on percussion of the tendo-Achillis whilst the leg is supported anteriorly near the foot, and flexed so as to be at right angles to the thigh, has been present in every normal individual in whom I have tried it (more than 200 of various ages). It is very readily obtained, even with the boot on.

The elbow phenomenon is also normal, and of 300 consecutive cases that I have tested, it was only absent in one, a man over six feet in height, and with very powerful and thick muscles, who could not be got to hold his arm in proper position and relax his muscles.

This phenomenon is obtained by pulling the arm a little away from the body, the physician standing behind the patient, placing his left hand under the lower end of the humerus and raising it to a horizontal position, letting the forearm hang vertically and quite loosely. Then the tendon of insertion of the triceps is struck just above the olecranon. As this tendon is very short, great care must be taken not to strike the belly of the muscle, because that will produce a contraction even when the tendo-muscular phenomenon is absent.

The wrist phenomenon is obtained by holding the lower end of the forearm in one hand, with the elbow bent at an angle of about 95°, and the hand hanging quite loosely, then striking

the tendon of the extensor on the radial side of the wrist. The biceps contracts and causes flexion of the forearm on the arm, and sometimes the extensors contract and cause extension of the hand.

The wrist phenomenon is much more often absent than present. In the cases (more than 200) in which I tried it, it was present in twenty, most of whom were young women. Dr. Buzzard tells me he has found it in much older people also, both men and women. Its absence, however, does not mean anything, but when exaggerated it is often useful, more especially as it can be so easily tried.

The reactions of the zygomatic and other muscles are not of so much value. They are, as a rule, present normally, but are very different in different individuals.

These tendo-muscular phenomena may be :

1. Absent.
2. Exaggerated.
1. They are absent in the following diseases :
 - a. Locomotor ataxy { Posterior root zones and sensory roots sclerosed.
 - b. Infantile paralysis.
 - c. Adult spinal paralysis.
 - d. General spinal paralysis.
 - e. Progressive muscular atrophy (some cases).
 - f. Diphtheritic paralysis (Ord), pathology unknown.
 - g. Pseudo-hypertrophic paralysis. { Disease in muscle. It will probably be found absent in other diseases of muscle.
 - h. General paralysis of the insane. { One case recorded by Westphal. There was sclerosis of the posterior root zones, but no ataxia.
 - i. Lesions of sensory nerves.
 - j. Lesions of motor nerves.
 - k. Loss of tone in the muscle from any other cause, such as fatigue or overwork. It is for this reason, I believe, that it is absent just after an epileptic fit; and if the reason I have given be correct, it

will be found absent after any severe convulsion, whatever be the cause of the fit. Poisoning by curare completely abolishes the phenomenon.

The accompanying diagram shows the various parts of the cord which are affected in different diseases producing alterations in tendo-muscular phenomena.

The posterior root zones are affected in *tabes*; and in Westphal's case of general paralysis of the insane without tabetic symptoms the same parts were affected.

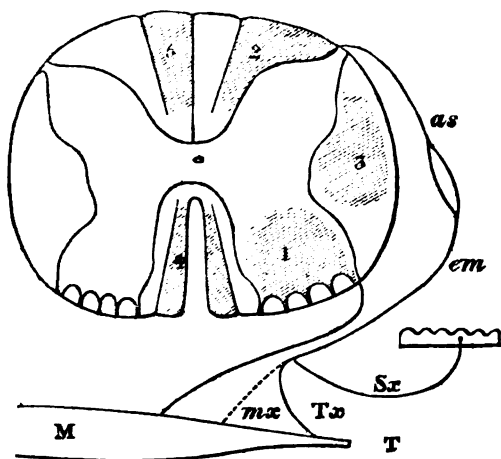


FIG. 2.—SECTION THROUGH SPINAL CORD, SHOWING PARTS AFFECTED IN VARIOUS DISEASES.

1. Anterior cornu. (*Infantile paralysis, &c.*)
2. Posterior root zone. (*Locomotor ataxy.*)
3. Crossed pyramidal tract in lateral column.
4. Direct pyramidal tract in anterior median column.
5. Posterior median column. (*Ascending sclerosis.*)

} Descending lateral sclerosis.

The afferent sensory nerve is represented below arising from the three different parts, skin, tendon, and muscle, *Sx*, *Tx*, *mx* (dotted line), respectively.

The anterior horns or cornua are affected in four diseases, *infantile paralysis, &c.* The muscle itself is affected in *pseudo-hypertrophic paralysis, exhaustion, &c.*

In all the above there is an absence of the phenomena. Presently, diseases will be described in which they are increased,

and in these the lateral columns will be found affected as shown in the diagram. And when the descending sclerosis is due to some lesion in the brain, it will be found that degeneration has taken place not only in the lateral column of the opposite side, corresponding to the fibres which cross over between the pyramid of the medulla oblongata and therefore called the "crossed pyramidal tract," but also in the anterior median columns of the same side as the lesion, in fibres which do not cross at the anterior pyramids and so called the "direct pyramidal tract." When sensory nerves are cut, the posterior median columns degenerate from below upwards; but the parts corresponding to the cord thus sclerosed are not affected as to the tendo-muscular phenomena.

The absence of the knee phenomenon in locomotor ataxia is one of the very earliest symptoms of that disease, and is often of service in diagnosis. Cases said to have been tabes have been recorded in which, so far from the knee phenomenon being absent, it has been exaggerated. Westphal says he has never met with such a case, and throws doubt on the diagnosis of those published. Of course there may be a stage in tabes antecedent to that in which the knee phenomenon is absent, but if there is it is so early, that there would be none of the other symptoms present, inco-ordination, shooting pains, amaurosis, small and often unequal pupils, which do not react to light, but contract on accommodation, &c.

The early failure of the knee phenomenon suggests that whenever a patient complains of defect of vision, or when his pupils are seen to be small, &c., tabes should be suspected, and the knee at once tested. If there is no reaction the disease is in all probability tabes. In diseases with subjective sensations which might give rise to suspicions of tabes, the presence of the knee phenomenon might decide the diagnosis.

From the foregoing remarks on the cause of the absence of the tendo-muscular phenomenon one can easily see its application in tabes. In this disease there is a chronic inflammation going on in the outer portion of the posterior columns of the spinal cord, abutting upon and at last involving the posterior grey horns and the posterior (sensory) roots just as they enter the cord. Chronic interstitial inflammation of the cord (sclerosis) is comparable with cirrhosis of the liver or granular kidney. The

part affected becomes harder than normal, and its proper tissue elements are destroyed and replaced by a fibroid tissue. In this way the reflex loop is destroyed at this portion of the cord. There is, as a consequence, a loss of tone in the muscles corresponding to the part affected, and there is also an absence of the tendo-muscular phenomenon.

Debove and Boudet, in the '*Archives de Neurologie*,' have shown by means of an instrument called the myophone, for measuring the tone of a muscle, that there is diminished tone in groups of muscles in locomotor ataxy and always in the quadriceps extensor. They very ingeniously account for the inco-ordination in this disease. They say that it requires more stimulus to cause an atonic muscle to act than a healthy one with tone, and that the voluntary stimuli sent down to the muscles of the extremities in ataxics, cause some muscles (tonic) to contract before others (atonic); hence the unsteadiness. There is an attempt to correct this by a maximum contraction of all the muscles involved, and hence the exaggerated muscular action, so characteristic of this disease.

The disease may affect any portion of the spinal cord, but for some hitherto unexplained reason it affects chiefly the lower part, and gradually diminishes towards the upper part. One finds, therefore, that in all cases the knee phenomenon and the ankle phenomenon (not ankle clonus, but contraction of the gastrocnemius to a blow on the tendo Achillis), are quite absent, whilst the elbow phenomenon is seldom absent.

The following is a case in point, and is interesting also, inasmuch as it is a case of locomotor ataxy in a woman, which is not very common.

Mary Ann D—, æt. 67, came under my care at the National Hospital for the Paralysed and Epileptic on the 18th October, 1880. For the last two or three years she had had shooting pains and soreness in the epigastrium, which had become much more severe the last few months. Then she found difficulty in walking about in the dark, and now can scarcely get along in the daytime.

Her gait as she entered the room was typically ataxic, and on examination it was found that she had numbness and anæsthesia over the front part of both legs and over both thighs (superficial to the quadriceps). No anæsthesia in the upper

extremities, and no shooting pains, and no inco-ordination in them. Complete absence of knee and ankle phenomena. Elbow phenomenon present. She had also dimness of vision and small pupils, which did not contract in the least to light, but a little to accommodation.

But the elbow phenomenon may be absent in locomotor ataxia, as the following case shows :

Thomas S—, æt. 40, came under my care at the National Hospital on the 28th of May, 1880. Has suffered for six or eight years with shooting pains and difficulty in walking, more especially in the dark. Has had syphilis.

Present condition.—Extreme form of ataxic gait. Cannot get along at all without assistance, and throws his legs about with great violence. Much difficulty in keeping upright. Considerable anæsthesia of feet, legs, and thighs, and also of arms, especially in the distribution of the ulnar nerve. Slight inco-ordination in upper extremities, as shown by the fact that he is unable to touch the end of his nose quickly. He nearly always misses it, and sometimes touches the malar bone or the lip. Pupils equal, do not contract to light, but slightly to accommodation.

Total absence of knee, ankle, and elbow phenomena. Direct percussion on the quadriceps, gastrocnemius, or triceps, causes contraction in each.

In all probability the posterior sclerosis has ascended as high as the cervical portion of the spinal cord, and thus is affecting his upper limbs.

In infantile paralysis the tendo-muscular phenomena are absent in the paralysed parts. This may be of use in diagnosis. I have seen a case in which the upper and lower extremities of one side were involved, and which was diagnosed as cerebral hemiplegia. In the latter, however, the knee and elbow phenomena are not abolished, as was the case in this instance. This indicated a spinal origin, and the subsequent history of the case, great muscular wasting, &c., proved it to be one of infantile paralysis. Whilst in another case, which was diagnosed as infantile paralysis in the upper and lower limbs of one side, I found increased tendo-muscular phenomena, and the case proved to be one of cerebral hemiplegia with descending sclerosis.

In adult spinal paralysis, that is, acute anterior cornual (or polio-) myelitis, the tendo-muscular phenomena are speedily abolished. Their absence is greatly in favour of its being an organic disease, whilst their presence would be conclusive against the above disease, at all events. Of course in this complaint there would be complete paralysis and no anæsthesia of the affected parts, and the muscles would rapidly waste. With the exception of the rapid and extreme wasting, hysteria may put on the form of anterior cornual myelitis, but in hysteria the tendo-muscular phenomena in the limbs affected would probably be present. Dr. Buzzard states that he has never yet seen them absent in hysteria.

In general spinal paralysis the phenomena would be absent or not according as the portion of cord corresponding to the origin of the motor nerve to the muscle was or was not affected.

Similarly in progressive muscular atrophy. In this disease the tendo-muscular phenomenon is sometimes exaggerated, but there is in such cases concomitant disease in the lateral column (Buzzard).

In diphtheritic paralysis the phenomena disappear, and return with returning voluntary power. Seeing that there is in these cases also anæsthesia it is probable that the diphtheritic lesion producing the paralysis is in the spinal cord.

In pseudo-hypertrophic paralysis the muscular fibres become destroyed, undergoing fatty degeneration, whilst there is a considerable development of connective fibrous tissue. Hence there can be no contraction of any kind. In his work on this disease Dr. Gowers states that it is diminished at first, and finally disappears. Probably in other diseases of muscle, such as inflammation, it will prove to be absent.

In a case of general paralysis of the insane, recorded by Westphal in the '*Berl. Klin. Woch.*,' there was sclerosis of the posterior root zones as in tabes, but no ataxia.

My colleague, Dr. Savage, tells me that it is the rule for the tendo-muscular phenomenon to be exaggerated in general paralysis unless there be ataxic symptoms in addition. I have examined twelve cases, some at the National Hospital, but most, with Dr. Savage's permission, at Bethlem Hospital. Of these it was exaggerated in five; normal or only slightly marked in three; absent in three, of whom one had slight ataxic symptoms,

not being able to stand with eyes closed and feet together, nor to turn suddenly when walking, &c.; the other two had no ataxic symptoms. The remaining case was that of a girl only twenty-one years of age, who had been an actress, in whom the symptoms of general paralysis of the insane were well marked, but she had no ataxia; the knee phenomenon was well marked in the left leg, but completely absent in the right.

Hence it appears that in general paralysis of the insane the tendo-muscular phenomena may be normal or even exaggerated, or may be absent; and when absent there may or may not be ataxic symptoms. In no case where the knee phenomenon was present were there ataxic symptoms.

Lesions of sensory nerves or motor nerves alone are not often met with because the spinal nerves are all mixed nerves beyond the junction of the posterior (sensory) and anterior (motor) roots, and the roots are seldom implicated. But when lesions do occur in either root or in a mixed nerve the phenomena are absent. Thus in meningitis which involves the roots, and in section of the anterior crural nerve (Erb) they are absent.

Lastly, that their absence may follow loss of tone in the muscle from any cause, is a generalised statement, which I believe will be found to be fully borne out, as our experience grows, which is supported by the facts already adduced, and which receives additional support from Westphal's observation, that the phenomenon is absent for a short time just after an epileptic fit.

2. The tendo-muscular phenomena may be exaggerated.

Before enumerating in what diseases they are exaggerated, a condition may be described closely allied to these phenomena, and generally found occurring where they are exaggerated. This is the so called clonus.

The best idea of clonus may be obtained from what might be called the natural ankle clonus. It must be in the experience of most, that when whilst seated the heel is raised, the toes still resting on the ground, the leg can be set into a rapid up and down movement by a series of alternate contractions and relaxations of the gastrocnemius and soleus, which though voluntary at first, go on quite involuntarily after the initial movement.

In certain diseases, if the lower end of the thigh be supported by one hand and the foot of the dangling limb be grasped by the other near the toes and raised so as to bring the leg half way or more towards complete extension and then suddenly flexed on to the leg, so as to put the gastrocnemius and soleus suddenly on the stretch, they are thrown into a similar series of alternate contractions and relaxations, so long as the toes are kept pressed towards the leg in front. This is called the "ankle clonus." It cannot be obtained in health in this way. A variety of it is obtained by twisting the foot inwards so as to stretch the peronei, which then are thrown into clonus.

A similar series of alternate contractions and relaxations can be obtained in the quadriceps extensor, by placing the lower extremity in perfect extension, the muscle being quite relaxed, so that the patella moves freely over the joint, and then exerting sudden tension upon the muscle and keeping it up, either by a pull upon the patella, or better (Gowers), by placing the finger above the patella and percussing it downwards and slightly backwards, keeping it well pressed against the upper edge of the patella. This is called the knee clonus.

There is a slow form of knee clonus obtained but very rarely, by percussing the ligamentum patellæ. The foot swings forwards and backwards like a pendulum (Gowers).

A similar clonus can be obtained in the great toe by suddenly extending it so as to stretch the plantar muscles. And one at the wrist, by suddenly extending the fingers and hand, so as to stretch the flexors. No clonus can be obtained at the elbow-joint. Hence the various kinds of clonus may be enumerated as

Knee clonus	{ quick.
	{ slow.
Ankle clonus	{ direct—gastrocnemius and soleus.
	{ lateral—peronei.

Toe clonus.

Wrist clonus.

In all these varieties, except the slow knee clonus, the movements are remarkably rapid and uniform. There are from 6 to 8 contractions per second, or 360 to 480 per minute, a rapidity almost too great to be counted, Dr. Gowers has,

however, made tracings on a revolving drum, the writing point being attached to the foot. He was then able to calculate the number per second, and to point out that the tracings were very regular, and that there is no complete relaxation between the contractions, a certain amount of 'residual contraction remaining between each,' which residual contraction is greater after several contractions. He says the clonic contractions are the result not of a reflex but of a direct stimulation of the muscle by the sudden tension or vibration, but that the muscle is in a state of irritability to such tension, and that this irritability is brought about by a reflex action through the cord, the afferent impulse arising in the muscular fibres by the sudden tension put upon them.

Whether this be true or not, the phenomenon cannot be produced unless the reflex loop between muscle and cord be present. The sudden flexion of the foot sets up the first contraction, and as the muscle is relaxing the flexion is kept up, and so starts another contraction, and so on, producing the clonus. But clonus cannot be obtained by a sudden flexion of the foot in a state of health, so that its presence indicates disease.

Another phenomenon is always obtainable where there is ankle clonus, and sometimes it comes on before the clonus in disease. This is the front tap contraction described by Gowers. It consists in flexing passively the foot on the leg so as to put the tendo Achillis and its muscles on the stretch, and percussing the muscles on the front of the leg (tibialis anticus, &c.). The vibration passes through to the gastrocnemius and soleus behind and causes them to contract. It is a single contraction, but it often sets up the clonus if the foot be kept flexed.

Increased tendo-muscular phenomena, front tap contraction and clonus, all occur in the same diseases. And whenever clonus can be obtained in a muscle the tendo-muscular phenomenon of that muscle will be exaggerated. For example, whenever there is ankle clonus a blow on the tendo Achillis will cause a much greater contraction of the gastrocnemius and soleus than is normal. But clonus cannot always be obtained where there is distinct and even great exaggeration of the tendo-muscular phenomena.

The diseases in which these exaggerations occur are :

a. Lateral sclerosis.

1. Primary or idiopathic.
2. Secondary to
 - Myelitis of cord.
 - Tumours of brain.
 - Hæmorrhage in brain.
 - Compression of cord.
 - Pott's disease.
 - Contusion of cord.
 - Disseminated sclerosis.

b. Chronic rheumatic affections. They are only rarely found in these affections, and in those cases where they can be obtained, there are other evidences of secondary changes in the spinal cord (Gowers).

c. Epilepsy, after the fit (Jackson).

d. Paralysis agitans (a few cases).

It thus appears that in all those cases whose pathology is known there is sclerosis in the lateral columns of the spinal cord (*vide* diagram, p. 65); and in the post-epileptic cases, Dr. Hughlings Jackson suggests that there is an exhaustion of the lateral columns.

The question at once arises, how does a lesion in this portion of the cord cause exaggerated tendo-muscular phenomena. Three theories have been offered.

1. *Irritation.*—It is said that the chronic inflammation (which constitutes sclerosis) of the lateral columns irritates and keeps up irritation in the anterior motor cells, and so increases the reflex irritability of the cord. The latter part assumes the truth of the reflex theory. If the atonic theory be the correct one, we might say that the irritation causes increased tone in the muscles, and so they more readily respond to local stimulation. That it does produce increased tone is shown by the fact that the muscles gradually contract more and more until rigidity supervenes.

2. *Removal of cerebral influence.*—Normally the brain is said to inhibit the reflex action of the spinal cord. Hence if its influence be removed, the reflex irritability of the cord would be greater. Under the same conditions the tone of the muscles would be increased.

3. *Unantagonised cerebellar action.*—Dr. Hughlings Jackson,

who proposes this theory, says that normally the cerebrum and cerebellum each has its own special influence on the cord, and that these influences antagonise each other: that in lateral sclerosis the cerebral influence is cut off, and thus the cerebellar influence, which is to cause contraction of muscles, is unopposed; and hence the phenomenon of increased irritability which finally goes on to rigidity.

The difficulty with regard to the last two theories is that neither the rigidity nor even the exaggerated tendo-muscular phenomenon comes on immediately when the cerebral influence is removed. But it must be remembered that a sudden brain lesion makes a deep impression on the spinal cord. I have seen a case of recent apoplexy with right hemiplegia in which there was complete absence both of superficial reflexes, and of the tendo-muscular phenomena, which gradually returned.

The important point to remember, however, is the fact that so far as pathology has thus gone, increase of the tendo-muscular phenomena, and *a fortiori*, clonus, which is a further development of the same, indicate lateral sclerosis of the spinal cord, *i.e.* sclerosis in the crossed pyramidal tracts (*vide* Fig. 2, p. 65); and the particular muscle affected will localise the spot in the cord. Thus, ankle clonus means lateral sclerosis in that part of cord which gives off the sacral nerves, whilst exaggerated knee phenomenon indicates sclerosis where the third and fourth lumbar nerves come off (anterior crural). Hence a transverse myelitis at the origin of the third and fourth lumbar nerves would cause abolition of the knee phenomenon; and if sclerosis descended from that myelitis, there would be increased ankle phenomena, and perhaps ankle clonus.

Theoretically anything that will increase the irritability of the muscle might cause increased tendo-muscular phenomena, or even clonus, and so irritation of sensory or motor nerves, or an irritable condition of the muscles themselves from disease, may possibly be discovered as causes of these phenomena as our knowledge advances.

The commonest cause met with is descending sclerosis in ordinary cerebral hemiplegia. Westphal has seen a case in which ankle clonus and increased knee phenomenon came on an hour after an attack of apoplexy; but this must be exceedingly rare. As a rule it takes more than a week, during which inflam-

matory changes are going on down the motor tract from the hæmorrhage in the brain.

Hence it is important in cases of hemiplegia to test these phenomena, for if they begin to be exaggerated, rigidity will probably supervene. Charcot says it will certainly come on, but there are exceptions. The following is a case of spinal curvature, probably with myelitis and descending sclerosis in consequence :

A. E. D—, girl, æt. 12, attending my out-patients' at the National Hospital since beginning of present year. She has had spinal curvature in the dorsal region for five or six years, with gradually increasing loss of power in the lower limbs. She cannot walk without assistance, and her feet are dragged along the ground.

Greatly increased tendo-muscular phenomena in both lower limbs, ankles and knees, and also ankle clonus and knee clonus. Elbow phenomena present but not exaggerated. Wrist phenomena absent. No anæsthesia.

The following is a case, also attending at the National Hospital, in which I believe there is lateral sclerosis, probably secondary to some cerebral mischief.

Alice T—, æt. 18, had convulsions soon after birth, and about the same time her head became very large. Has always been very slow intellectually, and her memory is bad. No weakness and no loss of sensibility. Head now measures twenty-four inches. She has rather the appearance of hydrocephalus, and there is slight external strabismus of the right eye; but when she is asked to follow some object the eye adjusts itself, and both eyes then follow the object quite normally. No diplopia. All the tendo-muscular phenomena well marked, including that of the wrist. Knee clonus on both sides. Superficial reflexes fairly well marked on both sides.

The following is a case of epilepsy, the patient working in lead as a painter. The tendo-muscular phenomena are much exaggerated, and it is a question whether they have any connection with the fits or the lead, or are quite independent.

James H—, æt. 34, subject to fits for eighteen years. Has always worked in lead and has had colic, and generally suffers from constipation. The fits last a few minutes, and he struggles

a great deal. His warning is a glimmer before the eyes, or a cutting pain in the stomach.

Knee, ankle, elbow, and wrist phenomena are greatly exaggerated on both sides. No clonus.

The following is an interesting case with wrist clonus, which is rare :

Louisa S—, æt. 45, came to my out-patients' at the National Hospital on the 8th November, 1880. She had rheumatic fever fourteen years ago. Eighteen months ago had an attack of right hemiplegia, with partial aphasia, the latter lasting several weeks.

After a time she recovered use in the right leg, but the hand has remained nearly useless. She can raise the right arm a little, but cannot flex the forearm on the arm, nor flex the fingers. There is a well marked thrill and a loud presystolic bruit.

All the tendo-muscular phenomena are greatly exaggerated on the right side, and better marked than normal on the left. Ankle, knee, and wrist clonus on the right side. The wrist clonus is obtained by taking hold of the patient's wrist with the left hand, and placing the tips of the fingers of the right hand under those of the patient's hand and suddenly raising them so as to extend the wrist, and put the flexors of the forearm on the stretch ; they begin to contract and relax rapidly (six per second), but soon stop. The wrist clonus is always obtainable when she first comes into the room ; but after trying it several times, it cannot be further obtained, for what reason is not clear. There is no clonus obtainable on the left side. The diagnosis here is cerebral embolism from mitral stenosis, with subsequent descending lateral sclerosis.

3. The special senses.

The reflex actions in connection with the special senses of smell, taste, and hearing are neither numerous nor important as aids in diagnosis. Exaggerated reflex symptoms may occur in connection with them. For instance, odours may cause vomiting, an attack of asthma, or fainting ; similarly, sounds sometimes cause syncope. But the most important nerve in respect

to reflex action in the diagnosis of disease is the optic. This results from the fact that the iris is visible, so that its movements can be seen through the transparent cornea.

The two sets of fibres, the radiating and the circular, of which the iris is composed, are of the involuntary kind, and are constantly in a state of slight tonic action, which is kept up by two separate nerves.

The tonic action of the circular fibres is kept up by a reflex process through the optic and the third nerves, and not by an automatic influence of the ganglionic centre of the latter nerve in the aqueduct of Sylvius. This is proved by the fact that when the optic nerve is cut the pupil dilates, and no further dilatation is produced by cutting the third nerve.

The tonic action of the radiating fibres is kept up by the sympathetic nerve, not by any reflex process so far as is known, but by an automatic influence constantly streaming from a dilating centre in the aqueduct of Sylvius (just behind the origin of the third nerve), down the spinal cord, and out at the lower cervical, and one or two upper dorsal nerves, and so into the sympathetic. When, therefore, the eyes are shaded or the eyelids closed, the tonic influence of the sympathetic is unopposed, and the pupils dilate. It is said that the sympathetic which supplies the radiating fibres of the iris does not pass through the lenticular ganglion but passes with the first division of the fifth nerve, and along its nasal branch, and thence through the long ciliary nerves. Moreover, the fifth nerve is said to have fibres in its first division, derived from the Gasserian ganglion, which are able to dilate the pupil independently of the sympathetic, though it is thought by some that these fibres of the fifth nerve act really on the blood-vessels of the iris and thus influence the pupil indirectly. The sympathetic acts directly on the radiating fibres, for it will act in a bloodless eye.

Whenever a sensory nerve is strongly irritated the pupils dilate. This occurs after excision of the superior cervical ganglion in an animal; from which Vulpian, who performed this experiment, argued that other nerves supply the radiating fibres of the iris, besides those which pass along the sympathetic.

But the dilatation of the pupil following strong stimulation

of a sensory nerve may be due to inhibition of the third nerve. To see if this were so or not, my colleague, Dr. Pye-Smith, has been kind enough to make the following experiment:

A large cat was placed under the influence of chloral (20 grains). Tracheotomy was performed and artificial respiration commenced.

The left superior cervical ganglion was next excised.

The left pupil became smaller than the right.

The right sciatic was now stimulated with progressively stronger currents of the Dubois-Reymond induction coil. With the secondary coil at 20, no effect was produced; at 15, still no effect; lastly, at 10, the strongest current employed, the right pupil dilated a little, the left remaining as before. The pupils responded freely to a considerable stimulus, but after being contracted by artificial light they both dilated on stimulating the right sciatic with the secondary coil at 10.

The skull was now opened and the left third nerve divided. A bright light produced contraction in the right pupil, but had no effect on the left.

The right sciatic nerve was again stimulated with the secondary coil at 12, 10, and 8 respectively, producing dilatation in the right pupil and no effect in the left in each case.

When allowed to remain undisturbed, under diffused light, the left pupil (third and sympathetic both divided) remained widely dilated, whilst the right pupil (no nerve divided) remained very contracted. Next, the distal end of the divided third nerve was stimulated, producing retraction and internal strabismus of the left eye, but no contraction of the pupil. (The heart was beating very feebly and the abdomen had already become cold.)

The above experiment shows that the fifth nerve has no fibres in it which cause dilatation of the pupil on strongly stimulating a sensory nerve, but that this result is brought about through the third nerve, probably by inhibiting the tonic influence kept up reflexly by this nerve upon the circular fibres of the iris.

The third nerve supplies both the circular fibres of the iris and the ciliary muscle, and when the latter acts during accommodation the former acts by association, and thus the pupil contracts.

The size of the pupil can be altered also by drugs, duboisia, atropia, physostigmin, &c.

Practically if the pupil responds to either light or accommodation it will also to drugs. But it may respond to light and not to accommodation or to accommodation and not to light, or to neither accommodation nor light; in the last case drugs may or may not produce an effect. Again, each eye must be examined separately and in combination with its fellow. If the left eye, for example, be closed, and the right eye does not now respond to light, but contracts on opening the left eye, it indicates lesion in the afferent sensory path of the right eye, which will be found to be blind. If one eye be blind from a lesion in the optic nerve the pupils of both eyes contract readily when light falls upon the sound eye.

Drugs are to be used when light and accommodation fail to produce any action. Atropine dilates and eserine contracts unless there be some disease in the iris itself, or some adhesion from previous iritis or glaucoma.

It would greatly facilitate matters if it could be stated that under a given degree of illumination the pupils would be a certain definite size, which could then be taken as a standard. But though this can not be done, considerable variety being met with in different individuals, yet certain generalised statements may be made. Under diffuse daylight with the accommodation relaxed the pupil averages $3\frac{1}{2}$ mm.¹

Such a pupil when the patient accommodates to a near object, say at a distance of two feet, contracts to 3 mm. In other words, in passing from the relaxed condition to one of accommodation the pupil contracts half a millimetre ($= \frac{1}{16}$ th inch). Much variation will be met with not only as to the amount of the diminution in size but also in the rapidity with which it takes place. It has been my impression, whilst examining a large number of cases, that it is more in quantity and greater in rapidity in young people than in old, in women than in men, in the weak than the strong.

The pupil is larger in dark than in light-coloured eyes.

¹ All measurements are taken by means of Nittleship's Pupilometer, which consists of a series of round black spots on a card, each spot numbered. Each number shows the diameter in millimetres, and the measurement is taken by comparing the spots with the pupil, the card being held flat with the iris, and close to the outer canthus.

If the pupils be closely watched whilst the patient is gazing on a fixed distant object, they will be observed to oscillate irregularly; and if by means of a pencil the contractions be represented on paper by an upstroke, and the dilatations by a downstroke, a tracing will be obtained showing a certain number of movements in a given time, say thirty seconds. If now the patient accommodates by looking at a near object, the pupils still oscillate irregularly, and if a tracing be taken, as before for thirty seconds, it will be found that the movements are not so frequent, nor are they so great. I have taken a great many tracings in this way, and have found that the oscillations in thirty seconds, when the accommodation is relaxed, are about twice as many as when the eye is accommodating for near vision.

The average number of oscillations during relaxation is about ten in thirty seconds, and about half that number during accommodation in the same number of seconds.

This phenomenon is quite absent in those cases of locomotor ataxy in which there is the Argyll-Robertson pupil (reaction to accommodation, not to light). In all probability, therefore, they are due to the varying degrees of light. They are not synchronous with the pulse nor with respiration, although slight movements have been said to occur with these (Foster's 'Physiology,' p. 467).

Dilated pupils.—The emotions, as is well known, will cause the pupils to dilate. In all probability they act through the sympathetic, for not only do we get accompanying vaso-motor phenomena (pallor of face) in many instances, but also if the corpora quadrigemina of an animal be stimulated after removal of the cerebrum, cries as of pain are produced, and the pupil dilates. It does not dilate, however, if the sympathetic nerve in the neck be previously cut.

Exhaustion causes dilatation of the pupils. They are larger in weak than in strong people. They are dilated during, and for some time after, a strong convulsive attack, as in epilepsy.

At the National Hospital for the Paralysed and Epileptic I have several times correctly inferred from the dilated pupils that a patient has just had a fit before coming into the room. They do not contract readily to light, and during the fit they are immovable to light. Dr. Gowers tells me that they

continue unresponsive to light for a short time after a fit. They are dilated during dyspnoea, partly, perhaps, through the muscular exertion which may end in a convulsion, and partly to the mental emotion. In other words, it is probably through the sympathetic. They react to light and accommodation in this condition.

The pupils generally become widely dilated at the point of death. They are nearly always larger than normal for some time after death, but in a few hours they have contracted so as to be of medium size. In some cases they remain dilated, especially if the eyelids are kept closed.

Mention has already been made of the fact that strong irritation of any sensory nerve will cause dilatation of the pupil, and reasons were adduced for believing that the result is produced partially, at least, by inhibition of the third nerve. Probably the sympathetic also acts directly on the radiating fibres, for there is often pallor of the face (vaso-motor phenomenon), produced by great suffering.

Any one who will take the trouble to watch the pupils whilst a tooth is being extracted, will notice the wide dilatation produced, especially if the operation be difficult and prolonged. I have watched it not only in these cases, but also in other painful operations, where the patients have refused anæsthetics, as in examinations of wounds and bruised limbs, the reduction of dislocations, &c.; and though a man may, by the powerful inhibiting influence of his will, restrain cries, muscular twitchings of face, and even the setting of his teeth, he cannot prevent his pupils dilating.

It is useful to remember this in cases of malingering. For patients will stand a great amount of pain, such as that produced by the electric battery, in order to keep up some feigned disease—for example, in railway injuries. Dilatation of the pupil, pallor of the face, or other vaso-motor disturbance produced by strong stimulation of a sensory nerve, tends strongly to prove that the patient can feel. It does not, however, absolutely prove it, because the lesion might be in the highest cerebral centres on the cortex of the brain, though there would in most cases be evidence of this.

Drugs will cause the pupils to dilate. Of these belladonna and its active principle atropia are the most important. The

ciliary muscle is at the same time paralysed, so that there is defect of vision from inability to accommodate. If there be hypermetropia amaurosis is much more readily induced, and a much smaller quantity of the drug suffices to induce it.

Other drugs, such as stramonium and hyoscyamus, also cause dilatation of the pupil, either locally applied or internally taken. They are not so extensively used as belladonna. Duboisia is now largely used; it is more powerful than atropia. It is thought that these mydriatics both paralyse the third nerve and stimulate the sympathetic. When fully dilated there is no reaction to light. In all cases where the pupils are dilated the possible use of some drug, such as belladonna, should be inquired into.

It is scarcely necessary to mention that adhesions of the iris are indicated when atropia produces no result.

Anæsthetics, such as chloroform, cause first contraction of the pupils, but if pushed too far the pupils dilate; hence the condition of the pupils is a valuable aid to the chloroformist.

From what has been already stated it is clear that disease affecting any part of the afferent sensory tract will cause dilatation of the pupils. Hence we find the pupils dilated behind opacities in the cornea, in front of opacities in the lens (*e.g.* cataract), or opacities in the vitreous humour, destructive lesions of retina, optic atrophy, optic neuritis when there is much affection of vision (although it is remarkable to what an extent optic neuritis may exist without causing dilatation of the pupils), and disease in the optic nerves, optic tracts, and corpora quadrigemina. The dilatation is due to the unopposed tonic action of the sympathetic. In all the cases vision is impaired or entirely lost.

They are dilated in effusions of blood within the cranium, especially when large, and more especially when in the ventricles. Dr. Wilks, in his work on 'Diseases of the Nervous System,' mentions the importance of remembering this, inasmuch as the patient, who is of course always unconscious with such a lesion, may be thought by the friends to be asleep; but the pupils are contracted in sleep, so that by raising the eyelids one can immediately detect the gravity of the case.

In ophthalmoplegia interna (paralysis of the ciliary muscle and the circular fibres of the iris) the pupils are dilated

and immovable to light, and the power of accommodation is lost. No lesion has hitherto been demonstrated post mortem in this disease, and it is curious that there are no less than three different theories as to the actual seat of disease. Mr. Hutchinson, who first described the affection, considered that the lesion would probably be found in the lenticular ganglion; he still holds that view. Mr. Hulke has brought forward quite recently, at the Ophthalmological Society, his opinion that it is in the peripheral endings of the ciliary nerves; whilst Dr. Gowers, in the debate which followed, expressed his conviction that it was at the central origin of the third nerve, somewhere near the aqueduct of Sylvius and corpora quadrigemina; and he brought forward several facts tending to support his view.

Cycloplegia or paralysis of the ciliary muscle is rare. There is no mydriasis, and the pupils react to light. It occurs sometimes after diseases producing great exhaustion, such as diphtheria. The cause is probably central (Hutchinson).

The external ocular muscles may be paralysed without any affection of the iris and ciliary muscle, and this condition is called ophthalmoplegia externa (Hutchinson).

These various affections, in which different parts of the third nerve are picked out as it were and the rest left intact, are probably due to central causes; and Dr. Allen Sturge suggests in his paper, read at the Ophthalmological Society, May, 1881, that there may not only be different nuclei of origin for different portions of the third nerve, but also higher co-ordinated centres which, when affected, would cause paralysis of the muscles acting together, such as the two superior recti.

Paralysis of the levator palpebræ and the external ocular muscles supplied by the third, together with a motionless and dilated pupil, and inability to accommodate, indicate a lesion in the trunks of the third nerve.

The pupils are dilated in protrusion of the eyeball. In a case of exophthalmic goitre, however, I found the pupils of normal size; they reacted to light and accommodation, and oscillated normally; they dilated on passing a strong faradic current through the neck.

The pupils are dilated when the aqueous humour is in excess.

Tobacco or its active principle causes dilatation of the pupil. They act to light and accommodation, but there is probably some dimness of vision. The "central region of the visual field is the part first affected, and remains throughout the seat of greatest relative defect; and, further, the periphery of the visual field remains of full size" (Nettleship, 'St. Thos. Hosp. Rep.,' vol. ix).

The pupils are not unfrequently dilated in typhoid fever, whilst they are contracted in typhus. Dr. Murchison says, however, that they may be contracted in typhoid, especially where there is great stupor and complete unconsciousness. There is anæmia as a rule with dilatation, hyperæmia with contraction. Indeed, in most cases of anæmia, as in aortic regurgitation and chlorosis, the pupils are large, and in hyperæmia, as in bronchitis or mitral regurgitation, they are small.

Contracted pupils.—Irritating lesions of the third nerves or paralytic lesions of the cervical sympathetics cause contracted pupils. Clinically such lesions are mostly met with on one side only; the pupil on the side affected is smaller than the other.

When in locomotor ataxy the pupils are contracted and do not react to light, they do not react to a strong stimulus passed through a sensory nerve (Erb); nor do they exhibit the variations in size met with in the normal condition. They respond to accommodation. Erb tries to explain these phenomena by supposing that the influence of the sympathetic is cut off by disease in the dilating centre in the aqueduct of Sylvius; or it may be in some part of the tract leading from this, along the spinal cord, as far as the second dorsal nerve. The absence of reaction to light is explained by another hypothetical lesion between the corpora quadrigemina and the origin of the third nerve in the aqueduct of Sylvius; as this cuts the reflex loop to the circular fibres of the iris, and there is no tonic action exerted by the third nerves except reflexly, the pupils ought to be of medium size when the accommodation is relaxed; the contracted state is not yet accounted for.

In some cases of locomotor ataxy the pupils dilate on strongly faradizing a sensory nerve. Dr. Gowers tells me that in such cases the pupils also react to light.

Another point, interesting on account of its relation to the sympathetic lesion, is that in nearly all cases of ataxy the pulse is quick and dicrotic. In most cases it will be found to be over 100 per minute.

It may be mentioned here that the pupils are contracted and the pulse quickened in poisoning by chloral hydrate, carbolic acid, chloroform (early stages), or opium.

During sleep the pupils are contracted and the pulse is slower than when awake.

In some cases of apoplexy, such as bleeding into the pons Varolii, the pupils are contracted and the pulse usually slow.

The pupils are smaller in old age, quite apart from any disease. They react both to light and accommodation, and are thus easily distinguished from the contracted pupils of tabes dorsalis.

The pupils are small in typhus fever (*vide ante*).

It has often been stated that the pupils are contracted in uræmic coma. Dr. Wilks (op. cit.) says they are not affected, and the comparatively few cases I have seen confirm the latter statement.

Unequal pupils.—Lesions of the cervical sympathetic of one side produce alteration in the pupil of the same side, generally causing contraction. This is met with in aneurism and tumours in the neck and thorax. The pupils react to light and accommodation. In some cases this inequality of the pupils aids the diagnosis of intrathoracic growth from valvular cardiac disease. Disease in the cilio-spinal region produces the same effect. The inequality is best seen in a dull light.

Lesions of the third nerve on one side produces inequality of the pupils. This condition is met with in tumours at the base of the brain and injuries to the skull. There is no reaction to light, nor to accommodation, on the affected side.

Both the third nerves may be affected in different degree, the pupils unequal and contracted or one dilated, as sometimes happens in tubercular meningitis. They may respond feebly to light or be motionless; the condition is one of some importance in diagnosing between this disease and typhoid fever.

Immobility of the pupils to light and to accommoda-

tion, with no paralysis of the muscles of the eyeball, may be due to adhesion of the iris from previous inflammation; the pupils on close examination are usually irregular, and atropine fails to produce dilatation.

A blow on the eye sometimes causes dilatation of the pupil, probably from paralysis of the circular fibres of the iris; the dilatation may last for months. At first there is no contraction to light, nor to accommodation; afterwards the pupil responds to both even before the normal size is regained.

Hæmorrhages into the brain or on the surface cause inequality of the pupils when the third nerve is affected. In acute disease of the brain there is inequality of the pupils, but no definite relation between the two has been established (Wilks).

In general paralysis of the insane the pupils vary. Most frequently they are both contracted either equally or varying in degree, or one pupil may be dilated, and the other normal in size or contracted. In cases where there are tabetic symptoms in addition, the pupils do not react to light, but contract to accommodation.

Through the kindness of my colleague, Dr. Savage, I have been enabled to examine several cases of general paralysis at Bethlem Hospital. I purposely took those cases with no symptoms of ataxia. In all the cases of undoubted general paralysis of the insane, the pupils were contracted to $2\frac{1}{2}$ millimètres; in most of them equally so, in a few they were unequal. They contracted still further on accommodation, but not at all to light. On passing a faradaic current through a sensory nerve (one electrode on the spine about the seventh cervical, and the other on the neck behind the middle of the sterno-mastoid), no dilatation was obtainable (the current was not very strong). The oscillations of the pupil which I have described were quite absent.

It would thus appear that the pupils in this disease are exactly the same as in locomotor ataxy. There is one point, however, that, if confirmed in a larger number of cases, may be useful in diagnosis. Mention has already been made of the fact that in locomotor ataxia the pulse is quick, generally over 100 per minute. On the other hand, in general paralysis of the insane, with the same condition of pupils, the pulse is as a rule not above normal, varying from 70 to 88. All the cases

examined were males, whose ages varied from thirty-eight to sixty.

In two cases the pupils were large. In one case, that of a man, thirty-three years of age, the left pupil was $=4\frac{1}{2}$ millimètres, the right $=3$ millimètres; both reacted to accommodation, but neither of them to light nor faradism. The patient got into a passion whilst under examination, and both pupils dilated. In the other case, a man, forty years of age, the left pupil was $=3\frac{1}{2}$ millimètres, the right one $=4\frac{1}{2}$ millimètres; both reacted during accommodation, but neither of them to light nor to faradism. The pulse in this case was 52 per minute.

Dr. Wilks (op. cit.) mentions the case of a man who insured his life and died about two years afterwards from general paralysis of the insane. It was found on inquiry that when the assurance was effected, the pupils were unequal, and he was suffering from general nervous debility attributed to over-study. This illustrates the importance of examining the pupils in all cases.

Mydriatics and myositics used on one side cause inequality. They may be used intentionally to deceive. There is usually defect of vision, and in some cases diplopia. There is no reaction to light, but in the contraction by eserine there is further contraction to accommodation. The inequality passes off in a day or two if all drugs be discontinued.

It has been said that in people subject to epilepsy the pupil of one side is larger than the other, the former being on the side which is most convulsed during the fits. Out of a large number of cases that I have examined on this point, in most the pupils were equal, and in the rest the larger pupil was as often on the side least convulsed as on the other. The pupils of epileptics have seemed to me rather larger than those of other people.

A SECOND CASE
OF
FRACTURE OF THE SKULL,
FOLLOWED BY
A COLLECTION OF CEREBRO-SPINAL FLUID
BENEATH THE SCALP.

By R. CLEMENT LUCAS, B.S.

In the 'Guy's Hospital Reports' for 1876 I have related the particulars of a case in which, after a severe simple fracture of the skull, a large collection of cerebro-spinal fluid appeared beneath the scalp, just above the left ear, in sufficient quantity to press down the pinna to a right angle with the head. The report is carried up to four months after the injury and two months after the patient left the hospital convalescent. I also refer in the paper to two similar cases which have been recorded: one mentioned by Erichsen as having occurred in a hydrocephalic child, and one more fully narrated, but deficient in post-mortem details, which was reported by Mr. Haward.

My report of the case was illustrated by a drawing of the tumour, and by a diagram of the fracture so far as it could be ascertained during life.

The patient was kept under observation, and one year and nine months after the injury she was seized with acute meningitis, which ended fatally in five days. Further details of this case, with a report of the very interesting post-mortem examination and a drawing of the brain, will be found in the 'Guy's Hospital Reports' for 1878.

It will be sufficient to state here that there was complete evidence that the cerebro-spinal tumour had communicated with the lateral ventricle, the descending and posterior cornua of which had become together dilated into a large cavity adherent to the skull.

The following case I am able to relate by the courtesy of my colleague, Mr. Howse, under whose care it was admitted a few days before he left London for the summer vacation in 1878. In his absence the patient fell to my charge, and I have ever since kept the child under observation.

R. P—, a boy, aged one year and eleven months, was admitted into the Evelina Hospital for Sick Children on July 24th, 1878. He had previously suffered from measles and pertussis, but not from scarlet fever.

About five o'clock in the afternoon he fell from a window, a distance of about ten feet from the ground, and pitched upon his head. He was picked up in a state of insensibility and brought to the hospital.

On admission he was quite unconscious, and remained in this condition throughout the night, but on the following morning he recovered his senses sufficiently to take notice of persons around him. His temperature at night was 99° . During the night there were frequent convulsive movements, and the child screamed at intervals. He has been sick directly after taking milk. He has passed urine twice, but his bowels have not acted. Temperature on the morning of the 25th, 101.3° , pulse 108, respirations 28. The whole of the forehead is much bruised and swollen, more especially on the left side. The left upper eyelid is extensively ecchymosed, of a deep claret colour, and too much distended to be raised. The right eyelid is slightly ecchymosed, and in this there is no subconjunctival ecchymosis. There has been no discharge of blood or fluid either from the ears or nostrils, and there is no paralysis. The anterior fontanelle is not closed, and the wrists are somewhat large. An ice-bag was ordered to be placed on the child's head, and three drachms of castor oil to be taken at once. Evening temperature the same— 101.3° .

July 26th. The child has not been sick since yesterday, and passed a quieter night, but is unconscious this morning. Pulse 76, not quite regular in rhythm. Bowels have acted once.

The right eyelid is more discoloured to day. Morning temperature 99° , evening 100° .

27th.—He has passed a quiet night. Bowels have not acted since yesterday. He has not spoken nor asked for his mother since the morning of the 25th. He takes milk well. Pulse 92, more regular. Morning temperature 99° , evening 100.4° .

28th.—About the same. Morning temperature 99.4° . In the evening it rose to 102.2° .

29th.—He is quite conscious. Pulse 96, regular. Morning temperature 99.5° , evening 99.2° . Swelling of forehead and eyelids subsiding.

30th.—Morning temperature 97.4° , evening 99.4° .

31st.—Hard ridges, indicating a line of fracture, can be felt on the forehead and scalp, extending back to the anterior fontanelle. The internal ridge is most elevated, and the external is apparently depressed. Pulse 96, not quite regular. Morning temperature 97.4° , evening 99.6° .

August 3rd.—The temperature during the last two days has been normal. He is progressing well, and sat up, for the first time to-day, in bed of his own accord. The swelling of the forehead and eyelids has been decreasing so that the ridges of bone can be more distinctly felt.

7th.—Has continued to improve gradually, his temperature having been during the past four days sometimes slightly below and sometimes a little above normal. His morning temperature to-day was 97.8° , but in the evening it ran up to 101.2° .

9th.—He is generally fretful, but has been gradually improving, and is able to talk to his mother. He sleeps well and his bowels act regularly. This morning it was noticed that the swelling on the forehead had increased in size, and it continued to increase during the day, becoming towards the evening of considerable size. The Registrar drew off with a hypodermic syringe about twenty minims of a clear faintly alkaline fluid, containing a few small flakes slightly tinged with blood. Yesterday the temperature was normal; to-day it was 98.5° in the morning, but rose at night to 101.6° .

10th.—To-day it has been noticed that the swelling pulsates. It is much smaller than yesterday, but it varies in size, increasing when the child screams. A ridge of bone can now be felt through the swelling, commencing about the middle of the left supra-

orbital ridge, and ascending irregularly on the forehead, somewhat to the right, as far as the scalp; it then crosses the median line and extends about half an inch to the right and beyond the anterior fontanelle. Another ridge can be felt, ascending at first vertically from the left orbit, then inclining slightly to the right to join the fontanelle on the left side. A long narrow depression can be felt between the ridges, broader above than below. Morning temperature was 99.4° , evening 100.4° .

11th.—The temperature taken this morning was 96.8° , and in the evening it was 97.8° .

14th.—During the last two days the temperature remained normal. This morning it was 98.8° , but in the evening it ran up to 103.4° , the highest temperature that has occurred since the accident. It was attributed to the excitement caused by the presence of visitors to-day.

15th.—The morning temperature was 98.4° , in the evening the temperature was 99.7° .

17th.—Yesterday the temperature was normal. This morning it was 97.8° , but in the evening it rose to 100.4° . The swelling of the scalp to-day has almost disappeared. The general condition of the child remains about the same.

30th.—The temperature has remained normal since the last report. The swelling of the scalp varies daily, but is never so great as formerly.

September 17th.—There has been nothing of importance to notice in the child's condition since the last report. The temperature has never, during the last month, reached 100° , even at night. The swelling varies from time to time, and is especially influenced by crying. A large gap is still to be felt in the frontal bone, extending from above the left orbit towards the fontanelle. The child is perfectly intelligent, and has a good appetite. He left the hospital to-day.

The child continued to attend among my out-patients for some months after it left the hospital, but there was nothing of importance to note in its condition. The mother received strict orders to bring it up immediately if seized with fits, sickness, or fever.

November 10th, 1879.—The child was brought up to see me, at my request, to-day. It is now a year and three months since the occurrence of the accident. The child has not

suffered from fits, nor from any symptoms that could be attributed to brain injury. He appears bright, intelligent, and in ordinary health. The mother says that the left eyelid swells when he is excited, and that it gives a heavy look to that eye, causing it to appear more closed than the other. He occasionally puts his hand to his head, and sometimes, but not often, complains of headache. There is no longer any swelling on the forehead, but a large fissure can still be plainly felt. This fissure commences above the left orbit, and after ascending a short distance curves slightly inwards, just internal to the frontal protuberance. It then continues to ascend upwards and inwards to the top of the forehead, lying to the left of the frontal fissure, which it afterwards appears to join, and so to be continued to the position of the anterior fontanelle, now closed. The fissure is widest at the upper part of the forehead, where it is full a quarter of an inch in width. It swells up slightly when he holds his breath and pulsation can be felt in it. The drawing indicating the direction and extent of the fissure was made at this time.

February 16th, 1881.—I have to-day again had an opportunity of examining the child's head. The fissure has not closed, and remains almost precisely in the same condition as when last reported. The edges of bone are probably united by fibrous tissue. He apparently suffers no inconvenience as a result of the accident, and is able to attend regularly at school. His friends state that at times they still notice a bulging over the line of fissure. It is now two years and seven months since the accident, but, after the experience of my last case, I cannot say that the report is complete. So severe an injury must be regarded as a permanent source of danger that may at any time determine an attack of acute meningitis.

Remarks.—The foregoing report adds a fourth case to those already recorded, in which, after a severe simple fracture of the vault of the skull, a large tumour of cerebro-spinal fluid appeared beneath the scalp; and I think there is now sufficient evidence to show that these cases belong to a very rare but distinct class of head injuries.

I would again point out, as I did when commenting upon my other case, that the patients who have suffered in this way have

all been young children. In Erichsen's case the age of the patient is not mentioned, but it is spoken of as a hydrocephalic child. Mr. Haward's patient was aged one year and seven months; the age of my first patient was two years and six months, and that of the case now reported was one year and eleven months at the time he met with the injury. It will be noted also that a fall from an upper storey was the cause of the fracture in each instance. Head injuries caused in this manner are anything but rare in adults among bricklayers, joiners, and such as are employed on scaffoldings or in the erection of buildings, but no case, so far as I am aware, has occurred in an adult of simple fracture followed by a large collection of cerebro-spinal fluid beneath the scalp. An easy deduction can be drawn from these facts—that the elasticity and thinness of children's skulls permit or determine the injury upon which this rare phenomenon depends, whereas in adults the strong and firmly ossified calvarium cannot be driven in to a like extent without an accompanying laceration of the scalp. Hence, similar injuries in adults are compound, and may be followed by the escape of cerebro-spinal fluid through the wound, but never, so far as recorded cases indicate, by collections beneath the scalp.

It is clear that in all such injuries the dura mater and the visceral arachnoid must have been lacerated, but is a wound stopping short at this point sufficient to cause so large an escape of fluid as has been observed in these cases? I think not; and I am inclined to believe that, had a careful post-mortem examination been possible in every case where the escape of clear fluid from the vault had been noted, some wound of the ventricular cavity would in each instance have been found.

The subarachnoid space, when irritated, rapidly pours out plastic lymph; and fluid in quantity on the hemispheres, though met with occasionally in association with atrophied brain, is not, I believe, found as a result of inflammation or after fractures. A slight wound of the ventricles, however, would allow of a very free escape of clear fluid, such as has been noted in several compound fractures of the vault.

The first case reported by me of a collection of cerebro-spinal fluid beneath the scalp was proved, by post-mortem examination

two years after, to have been caused by a wound of the descending horn of the lateral ventricle, and in the case now recorded, so similar in nature, I have little doubt that the anterior cornu was wounded. In both cases the injuries were so severe that laceration of the brain to the depth of the ventricle was not improbable.

In further support of the view that when large quantities of clear fluid escape through the vault the wound has reached the ventricle, I may again allude to the nine cases mentioned by Mr. Prescott Hewitt in his essay in Holmes' '*System of Surgery.*' It is not a little remarkable that seven of these cases recovered, so that the nature of the injury was merely a matter of conjecture, but in both the cases where a post-mortem examination was obtained, an aperture into the ventricle was discovered.

It is not necessary that the wound should, in the first instant, actually reach the ventricle, for it would appear that in the cases where post-mortem examinations were obtained, the outflow of clear fluid took place some weeks after the injury, in consequence of a softening process having extended to the ventricular cavity. Why, seeing that the visceral arachnoid must have been injured in these cases at the time of the accident, did not the fluid escape then or a few days after? Again, if a wound of the inner layer of arachnoid were sufficient to give rise to the escape of watery fluid, surely this would be much more frequently noticed. Why is it not met with in every case of punctured fracture when the brain is wounded by the fragments or by the foreign body? For instance, in the '*Clinical Society's Transactions*' for 1879, I reported a bullet-wound of the skull where the internal table had been driven back so as to perforate the dura mater and injure the brain. Why, in this case, did not cerebro-spinal fluid escape during the five days the man lived after the injury? It may be premature to state that in every case where watery fluid escapes from the vertex the ventricular cavity has been laid open, but all post-mortem evidence seems to point to this conclusion, whilst the view that the fluid escapes from the subarachnoid space rests only upon conjecture.



A CASE OF OSTEITIS DEFORMANS.

By C. J. SYMONDS, M.S.

As examples of this disease are not of frequent occurrence, and as the present one exhibits a striking malformation of one of the bones of the forearm, it has been thought worthy of a place in these Reports. Mr. Bryant has recorded a case in the volume for 1877.

The patient, Mrs. W—, whom I have had under observation for six years, is now sixty-nine years old. Excepting her father, no member of her family has, so far as she knows, suffered from any special disease. Her own three daughters are in good health, and except with these, she has never been pregnant. Her father suffered from “rheumatic gout,” but lived to the age of eighty, his hands were much deformed, and he was lame. Mrs. W— is quite sure that there was no bowing of the legs, and that her father’s complaint was entirely different from her own; indeed, her description of the malady justifies the use of the term employed by her. There is especially no history of tumour or syphilis, either in the patient or her family.

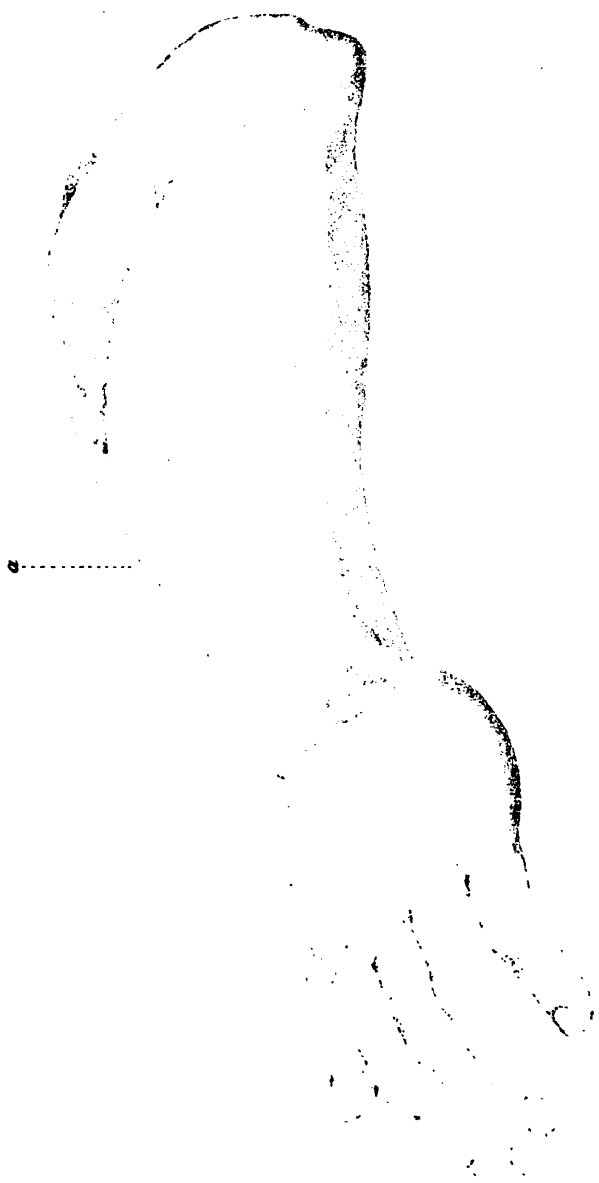
When about the age of forty-eight, without suffering any previous pain, and without any assignable cause, the left shin began to bulge forward. Under medical advice iodine was applied, but as this produced considerable pain it was discontinued. Within a short period the right shin began to acquire

the same appearance. The gradual increase of the deformity continued to be the only symptom till about twelve years ago, when she began to experience aching pain in the right arm, chiefly from the elbow to the wrist. This was frequently so severe at night, that rest was only obtained by hanging the arm over the edge of the bed. At that time she was occupied in household work, and did besides a good deal of sewing.

Eight or nine years after the bowing was first noticed in the legs, and soon after the aching pain began in the right arm, she noticed that the left arm began to "crook," and its movements to be impaired. Since this time the curvature of the radius has increased, and the bone has gradually assumed the position it at present occupies, without ever causing her more than an occasional ache.

Coincident with the pain in the right and the bowing of the left arm, the right hip became painful and prominent, and she states that even before this, her friends observed a limp in her gait. On inquiring more particularly into the amount of pain experienced in these several situations, she states that in the right arm it was so severe as to render her insensible to slighter degrees. Five years ago, while undertaking the duties of a cook, an ulcer formed on the left shin; this has increased continually, and is now her most distressing complaint. She is unable to remember exactly when the pain disappeared from the right arm, but it has ceased to attract attention since the formation of the ulcer. This arm is now free from pain, is strong, and exhibits no deformity whatever. The course of the disease has not been marked by any special feature, nor has any other joint become involved. For the last few years she has had occasional attacks of abdominal pain, accompanied by sickness, and has one or twice been very ill. At present there is nothing to indicate the nature of these attacks, the bowels act regularly, and nothing abnormal can be discovered in the abdominal viscera.

At the present time (December, 1880) Mrs. W— is in fairly good health, and is still possessed of considerable energy. She recently made a journey to Swindon, and is only prevented from being more active by the pain and inconvenience of the ulcer.



The disease is limited at present to the bones of the lower extremities and to the left radius.

Both tibiae are extensively thickened and bowed forward, while the fibulae remain unaltered. The whole appearance corresponds exactly with the illustrations accompanying Sir James Paget's paper in the 'Med. Chir. Trans.' vol. lx, pl. i, so that further description seems unnecessary. The lower two thirds of the left femur present the same form of enlargement, the thickening gradually diminishing upwards. This bone is slightly curved forward, so that when the limb is extended, while the patient is recumbent, the heel and upper part of the thigh are the only parts in contact with the horizontal. There is no sudden or bossy enlargement of these bones, the surface feeling only a little rough.

The joints of this limb, with the exception of the ankle—the movement of which is impaired by the oedema surrounding the ulcer—enjoy the full range of motion. The disease in the right femur is confined to its upper extremity. The trochanter is widened and its outline is ill-defined, but it is not more prominent than that of the other side. The limb is permanently rotated outwards, abduction in any position is impossible, and only slight adduction is permitted, while flexion to a right angle is free and unaccompanied by grating or pain. There is neither in this nor in any other joint further evidence of rheumatic arthritis. The right leg is five eighths of an inch shorter than the left.

The deformity which is the special feature in this case exists in the left radius, and is fairly well shown in the accompanying drawing. The bone is enlarged and elongated; it measures $9\frac{1}{4}$ ths inches in length over the curve, while the right is $9\frac{3}{4}$ ths. It presents posteriorly a conspicuous curve, gradually increasing in prominence to about the junction of the lower and middle thirds, where it ends in a sharp ridge, round which curve the extensors of the thumb, the ulna remaining unaltered. The radius curves also forwards, turning the hand into a position of complete pronation, so that the rotatory movements of the forearm are annulled. The extensor muscles form a slight rounded prominence in the middle third, having fallen towards the ulna, owing to the backward curve of the radius.

She is not aware that there has been any increase in the size of her head, but for future observation the following measurements were taken :

Circumference at level of middle of temporal fossa 21 in.

From occipital spine to base of nasal bones 14 in.

From mastoid process to mastoid process $9\frac{1}{4}$ in.

There is no curvature or rigidity of the spine. There is an old united fracture of the right clavicle.

ON THE RHEUMATIC DIATHESIS IN CHILDHOOD.

By JAMES F. GOODHART, M.D.

THE following paper has in it little that is novel. On the contrary, I would bespeak the attention of my readers, because the subject is one with which they must be quite familiar.

Diatheses, whatever may have been thought in times gone by, are not allowed to pass unchallenged now. But whether diatheses are realities, and whether there is such a thing as a rheumatic diathesis or not, are questions which involve far-reaching issues.

For some years past I have been in the habit of making careful inquiries both in the out-patient room at Guy's Hospital and also in that of the Evelina Hospital for Children upon the parental sick bill of the children that have been brought to me. The facts that have been collected form the basis of this paper.

I have no intention of concerning myself with the facts which others have collected, I had almost said times already without number. I am not unmindful that the subject has undergone repeated discussion, but were I to attempt to treat the question historically this volume would, I fear, not be able to contain the things that might be written. I shall only permit myself to say *in limine* as an index and reminder to those interested in the subject, and because articles in volumes of this kind are often forgotten or fall unheeded, that the 'Guy's Hos-

pital Reports' have already contained matter bearing upon one or other branch of the rheumatic diathesis, by Dr. Hughes, in a "Digest of 100 Cases of Chorea," in Series 2, vol. iv, and by Dr. Pye-Smith, in an "Analysis of Cases of Rheumatism, &c.," in Series 3, vol. xix. Dr. Pye-Smith, indeed, does not allow that there is such a thing as a rheumatic diathesis, but he shows that rheumatism is transmitted from one generation to another in 23 per cent. of the cases, and hereditary transmission embodies a large part, though not the whole, of the idea which the term diathesis expresses. And I suspect that any differences of opinion which we may seem to entertain are more questions of the meaning to be attached to words than of the essential difference of things.

After this it may appear to some that the proper point of departure for any observations I may have to make would be to define the limits of the term "diathesis." But definitions, while they have much to recommend them, have, on the other hand, an obvious disadvantage, that while they seem to clear the way, they often open up pitfalls which are very difficult, if not impossible to avoid. In the first place, the views of the writer must be accommodated, which may or may not be a difficult matter; and next the many points from which others may advance upon the subject must all be included—a task which is very certain to be one of considerable difficulty. Definitions for some must be comprehensive, for others exact. But the varying combinations which disease effects with constitutional build, in the changeful circumstances of social life, if they allow of the one, almost exclude the possibility of the other. And with reference to the term diathesis in particular, it seems to me that to impose upon it the restraints of an ill-fitting definition is to do the subject but questionable service; and to invite the criticism so rife just now that it has no meaning, I would say of it just what Dean Church says of civilisation—"It is, we all know, a vague and elastic word . . . but it expresses a substantial idea, it marks a real difference in what men are and can be."¹

Now, in very general terms my idea of diathesis is this: a something represented by a certain bodily conformation, which

¹ 'The Gifts of Civilisation.' By R. W. Church, M.A., D.C.L., Dean of St. Paul's,

is characteristic of its parentage; is possessed of a vital machinery which tends to work in a characteristic way; and which in turn tends to transmit its methods of working to its progeny. Diathesis, therefore, seems to me to be equivalent to family strain, and so reading it, I should call hereditary genius a diathesis, or rather a manifestation of one. The bodily form of the individual is often a re-embodiment of its antecedents, and its modes of working are characteristic.

The "gentle life" which comes of good breeding, the Bohemianism apparently so ineradicable in many, even though freedom be sought carefully and with tears, are equally manifestations of a diathesis as much as are the qualities of a thoroughbred horse. Take any one of these substantive expressions of a diathesis, and it could be said of it, with tolerable accuracy, it comes of such a stock, and that it is liable to evolve in its working certain characteristic phenomena normal or abnormal.

But I should not, as some would do, call such a disease as syphilis diathetic for many reasons, but chiefly because the disease is easily acquired; when transmitted from parent to offspring it wears itself out early in the second generation, and transmission to a third generation is not known.

In this sense, I propose to consider the rheumatic diathesis in children in the present paper. There are certain conditions or states in children—I do not by any means exclude adults from participation, but that I am not now concerned with them—which are found in association with a rheumatic strain. These are acute rheumatism, heart disease, chorea, headache, night terrors, what I shall call crises gastriques, nocturnal incontinence of urine, some forms of muscular spasm, nervousness, &c.

For the last three or four years I have been collecting evidence upon the question of the liabilities incurred by children of rheumatic parentage. Let us see with what results.

I will first say a word on the *intensity* of the rheumatic strain. Various opinions are held upon this question, but upon the whole it appears that authorities are agreed that rheumatism is capable of hereditary transmission, though not so strongly as many other diseases. From a large number of inquiries upon this point, however, it appears to me that there are few diseases which run more in families than it. The results are,

I grant, often less striking than those of phthisis, for instance, to take, perhaps, the disease which all would grant is one of the most hereditary. But this is because phthisis will kill off a whole family one after the other, and such a devastation as that it is impossible to ignore. Rheumatism, on the other hand, while tainting all, will only maim or destroy, perhaps, one; and the lesser rheumatic ailments are apt to elude notice. But examples of rheumatic family tendency might be quoted quite as marked as any consumptive one. For instance, a mother who says that she has had rheumatism in her knees, whose own mother has "rheumatic gout," and whose husband has had rheumatic fever, has, at various times, brought her entire family of six children to me. The eldest, a boy of fifteen, has had rheumatic fever twice, and has mitral regurgitation. The second boy, aged ten, has had rheumatic fever twice, and has also mitral disease. The third, a girl aged eight, had mitral disease, and died in the hospital. The fourth, aged seven, had rheumatic fever after scarlatina, and at the end of two years, through which I watched her, there was good evidence of mitral thickening progressing towards constriction of the orifice. The pulse was irregular, the first sound rough, the second persistently accentuated, and the impulse forcible. The fifth, a boy, aged four, was laid up all one winter with rheumatism. And the sixth alone gave no evidence of rheumatism, a fact which perhaps counts for less when I say that it died at the age of fourteen months of tubercular meningitis. The history of this one family would alone be enough to make one think very respectfully of the rheumatic diathesis and of the persistence of the rheumatic strain. Other facts all tell in the same direction.

Acute rheumatism in children is in the majority of cases inherited. I have notes of forty-four cases, twenty-six girls and eighteen boys. Eighteen had what I shall venture to call a good family history. Seven more a moderately good one. In four the history of rheumatism was indifferent. In nine there was no history, and in six others the history is not stated. Two thirds, therefore, have rheumatic antecedents of some sort.

What I have called good, moderate, and indifferent histories of rheumatism may be seen by a glance at the enumeration of

cases given in an appendix at the end of the paper. The prefix G. corresponding to good, M. to moderate, I. to indifferent; R. stands for rheumatic fever in the individual only; — indicates that there is no definite note on the subject in my notes; N. indicates that no history could be obtained. I may, however, say at once that a history of rheumatic fever in the father or mother, or brothers or sisters, has been called good. A history of rheumatic fever or rheumatic gout in relatives other than the immediate progenitors counts for moderate, unless, as in one or two cases, there is a clear history of rheumatic fever in more than one uncle or aunt.

I am by no means clear that in the group of moderates I have not underrated the intensity of the family strain. This is the group which includes grandparents, and it appears to me that there is a rather frequent tendency towards atavism in the transmission of rheumatism; and the occurrence of atavism, if frequent, would be a strong point in favour of the propagation of the disease in conformity with the ordinary laws of hereditary transmission.

Under indifferent come all things denominated "rheumatism." But for this group I wish to say that some of these are probably rheumatic fever cases, which in the hurry of note taking have been styled thus ambiguously. Histories of vague rheumatic pains have been rejected as meaning nothing, and where I call a complaint rheumatism it generally means some definite illness, though not long enough or definite enough to go as rheumatic fever. I must also say that as far as I have been able to keep to a strict use of terms, "Rheumatic fever" means usually in my notes an illness which has laid up the patient or relative for several weeks in bed.

It is necessary to say all this, because such statistics as these are often met by the objection that "rheumatism" means nothing. By excluding slight cases I have taken all possible care that rheumatism shall stand for a definite illness.

I come next to *heart disease* in children. I have notes of 137 cases, all of which have come under my own care. Following the same lines as those laid down for acute rheumatism, I find that forty-six cases had a good family history, ten a moderate, and seventeen an indifferent one, and in nineteen others the patient had had rheumatic fever. So that

ninety-two patients out of 137, or two thirds of the whole, were rheumatic, and most of them rheumatic beyond any doubt.

I shall assume, after what has gone before, that of the nineteen patients who had had rheumatic fever two thirds would have a family history of rheumatism, when the percentage of cases which give a family history would be only very slightly lowered, being something under two thirds of all the cases.

I have further divided the 137 cases more critically into two groups of rheumatic heart disease and non-rheumatic, which makes the rheumatic group somewhat larger. That is to say, ninety-six instead of ninety-two, because there are four cases which although wanting in definite history were rheumatic in all probability. Thus, one girl was subject to rheumatic pains, but had never been laid up, an amount of rheumatism quite sufficient in childhood to explain heart disease. Another child has a history of aching in the limbs, &c., and so on.

It is, however, necessary to say that in sixty-seven cases of rheumatic heart disease in adults there is less evidence of this persistent strain, but in adults the family tendencies are oftentimes forgotten, and the history is, on the whole, less reliable. It is obvious that in dealing with adults inquiries of this sort often come new to them, particularly in the lower classes, and at a time when, their parents being in many cases dead, the opportunity for gaining information has passed away. With children this is not so. In the majority of cases they are brought by the mother or some near relation, and two generations are within reach of inquiry which can give reliable results.

An analysis of the sixty-seven cases in adults shows that twenty gave a family history of rheumatism, twenty-two could give none, and in twenty-five no mention is made of the point.

Now, if two thirds of the cases of acute rheumatism give a history of hereditary transmission of that disease, and if nearly two thirds of all the cases of heart disease show similar antecedents, I think the facts are sufficient to justify the conclusion, not only that rheumatism is capable of transmission—for that is already allowed by most men—but that it will be transmitted twice in every three cases, and that it is therefore

a very pronounced strain. I think there can be no doubt of this, but I do not think it is acted upon in practice. Let me take an instance. Life insurance offices guard their policies most strictly against anything of the nature of phthisis, while there is not in the papers of some offices a single question devoted to ascertaining the existence or not of a family taint of rheumatism. I remember well looking over the papers of one who proposed to insure his life in one of the well-established offices of London. There was a strong rheumatic family history, and I searched in vain for any indication that this was regarded with any suspicion by the office in question. Yet I maintain that it is only proper and prudent to put an increased risk on such a life.

Acute rheumatism is transmitted as acute rheumatism, if I may so express myself, with the accompanying disease of the heart in many cases. Witness the forty-six cases of acute rheumatism with family history in two thirds of them, and the fact that forty out of ninety-six cases of rheumatic heart disease were auto-rheumatic. But this is by no means always the case, and it is the varieties of the rheumatic manifestation which seem to me to be of special interest, and to which I would particularly draw attention.

In the first place, a rheumatic mother may transmit something to the fœtus which leads to congenital heart disease—at least, this seems to me very probable from the following cases.

A mother had had rheumatic fever seven times (so she stated), and her child was born cyanosed and with a loud systolic bruit over the pulmonary area, leading one to think that there was probably some pulmonary stenosis with possibly a deficient ventricular septum. The child was still alive when I last heard of it, so that there has been no opportunity of verifying the diagnosis. In a second case the father had had rheumatic fever, and there was extreme cyanosis in the child, a systolic bruit at the apex and in the axilla, another over the mid-sternum, and another over the aortic valves. The nature of this case is uncertain, but it is probable that both mitral and tricuspid thickening existed, and probably either aortic or pulmonary stenosis also.

A third case has lately come under my notice. An infant of two months old was brought to me in an extremely wasted state

and very anæmic,³ with the statement that it had been so since birth. The action of the heart was rapid, and there was a loud systolic bruit heard all over the præcordial area. It was, however, more intense at the apex, and was heard loudly in the axilla and back. There was no thrill either at the apex or base, and nothing that I could detect abnormal in the tricuspid region. The case was, I think, most probably one of mitral regurgitation. On questioning the mother upon her previous illnesses, she told me that she had suffered from some very severe fever when she was twelve or thirteen years old, which had kept her to bed many weeks, and in which her chief symptoms were great pain, swelling in the joints, and much sweating and difficulty of breathing. The difficulty of getting her breath she made the most of, indeed, confessed to spontaneously; the other symptoms were only elicited by leading questions, but she was equally decided about them when they were explained to her. I therefore think it probable that her illness was an attack of acute rheumatism, and that the child was suffering from a rheumatic endocarditis of congenital origin.

This question of congenital rheumatic endocarditis has often been discussed, but whether or not such a condition occurs is certainly not yet one of the settled questions of pathology. It is not pretended that these cases are decisive, but they are to my mind strikingly suggestive. From this point of view I am somewhat doubtful concerning the unconditional truth of Dr. Bedford Fenwick's contention that cases of tricuspid stenosis are acquired and not, as usually held, congenital. I have, though not a believer in many conditions often considered to be congenital, always considered that when we find that mitral and tricuspid stenosis coexist the disease is probably congenital. I am still inclined to adhere to that opinion for many reasons, but chiefly in this place because I believe that rheumatic endocarditis may be initiated *in utero*, and when initiated any subsequent attacks of acute rheumatism in extra-uterine life, by intensifying the cardiac disease, would in many cases appear, judging only from symptoms, to be its cause.

A propos of this opinion, let me next say that I think there can be little doubt that a rheumatic parent may transmit a something to the child which may, in the course of years, pro-

duce definite valvular disease—a tendency to chronic endocarditis; I do not suppose that the valves are diseased at birth. But there is evidence to show that children of rheumatic parentage are liable to become affected by permanent heart disease without having undergone the process of acute rheumatism. For instance, only the other day this case occurred to me:—A mother brought her boy, of about nine, for chorea, which he had had repeatedly. I inquired for rheumatism in father or mother, but unsuccessfully. But noticing that the mother was extremely anæmic, I questioned her concerning her relatives, and found that the maternal grandmother had had rheumatic fever twice, and had been laid up six weeks each time. The mother had never had it to her knowledge, but I examined her heart and found a diffused impulse, thick grating first sound, with a short systolic bruit under the nipple; the impression given by the physical signs being that the mitral valve was thick and probably small.

These cases are usually explained by the assertion, which is no doubt correct in great measure, that acute rheumatism is very obscure in childhood from the mildness of its attack, and that any child may have had it without notice being attracted by any appearance of illness. A child complains of aching in its limbs, perhaps, nothing more, and is afterwards found to have heart disease. The interpretation put upon the fact by many is that the passing pains were due to the rheumatic fever of childhood, and the heart disease is at once the result and the evidence of this. It may, perhaps, be thought a mere question of words, but I think it better not to call these obscure pains which children suffer—quite insufficient in many cases to send them to bed—acute rheumatism. It is far better, I think, to say that the rheumatic taint leads to such symptoms, and in the same way as it causes chronic changes in the joints so it may lead to chronic changes in the endocardium, no acute endocarditis having been at any time present. Better far than to call a disease acute which has certainly never been so in any ordinary acceptance of the term acuteness, in order to square with the orthodox views of the causation of heart disease. I am inclined to think, too, that the parallel I would draw between the chronic joint affection and the chronic endocarditis is closer than I have here stated it to be. Some years

ago, when more in the surgical wards than I am now, I made inquiries concerning the family history of a good many cases of disease of the knee-joint, and I was surprised as well as interested to find that not a few patients so affected came of rheumatic parentage, yet in hardly any was there a history of previous acute rheumatism.

I might, again, instance a case which occurred to me only the other day. I was asked by one of my colleagues to see a girl who had obstinate unilateral headache. I found that she was suffering from a synovitis of the knee of some months' duration, and her dark hair and extreme pallor at once suggested rheumatic tendencies to my mind. I inquired of her as to acute rheumatism, and she told me that till the attack of synovitis after exposure she had never had an ache or a pain to her remembrance. But, on the other hand, her father had died of heart disease, a comparatively young man, just over forty, when she was an infant; and one of her sisters had been in the hospital with rheumatic fever. Had I not strong reasons for considering that she was an example of the rheumatic diathesis which was working in her knee and now in her head?

In former years it was taught that rheumatic synovitis differed from other forms of inflammation in being but rarely destructive, but in the widening knowledge which we are now acquiring on the subject of acute and chronic destructive joint disease in acute and chronic nervous disorders, that proposition will probably in the future fail to be established; and although being in many cases an acute disease in a previously healthy structure, rheumatic synovitis, like other acute inflammation, will probably in a large proportion of cases completely resolve, yet rheumatism will have to take its place as a definite cause of *chronic* joint disease, if not through acute synovitis, by the not less sure process of chronic thickening. And, to return to the organ more immediately under discussion, I believe it to be essential to the correct estimate of the various causes of valvular disease of the heart that we should learn to recognise more fully than is yet done the varied conditions which are liable to engender chronic hypertrophic or inflammatory changes in the valves. Here is one. The existence of mitral disease in a child is often the only evidence of a rheumatic strain apart from family history. A fair proportion of cases of

mitral stenosis in adults are to be explained in this way, unless we take up the view held by some, which I do not think is tenable, that they are of congenital origin.

I will take this opportunity of saying a word or two more upon this question of the congenital origin of mitral stenosis. I have alluded to it elsewhere,¹ but the fact with which I am dealing now allows of its reintroduction. I just now spoke of the probable occurrence of intra-uterine endocarditis. I think it probable that given such a condition children may not infrequently be born with some defect of the valves of this sort, which may be slowly accentuated in after life either by other attacks of rheumatism or by the ordinary wear and tear of a damaged and imperfect valve.

Dr. Peacock has ably contended for the frequency of valvular disease produced in this way, with this difference only that for intra uterine inflammation he would substitute *malformation*. To the extent I have indicated I am prepared to admit the existence of congenital disease, but I am not prepared to admit that mitral stenosis is congenital as mitral stenosis except in a small proportion of cases. And the ground of my objection is, I believe, unanswerable. It is this that in all my cases of heart disease in children assured mitral contraction is rare, and even doubtful cases are but a small proportion of the whole. The figures are as follows :

Rheumatic heart disease	96
Non-rheumatic „	41
Heart disease in the course of acute rheumatism	23
Choreic heart disease	6
	<hr/>
	166 ²

Of these cases five only were certainly cases of mitral contraction, judged, that is to say, by præ systolic or diastolic bruit and thrill; seventeen others have a ? against them. The valve was probably thick, but the physical signs of contraction were not distinctive. Some had a hoarse first sound, others an occasional slight thrill, and so on. The remainder, with the exception of eight cases which were cases of aortic disease, were

¹ "On Anæmia as a Cause of Valvular Disease," 'Lancet,' vol. i, 1880.

² In order to avoid any confusion of figures I have kept the individual groups of cases distinct, both in what has gone before and in that which follows.

examples either of mitral regurgitation (114 cases) or of alterations in quality of the first sound with displacement of the impulse, &c. Now, I think these facts admit of but one interpretation, both for constriction of the mitral and also of the aortic orifice, viz. that pronounced congenital imperfections can only exist in a small proportion of cases. Otherwise we ought to detect more indications, even if slight, of the existence of disease in early childhood than we do. But, as a fact, mitral regurgitation is the common form of heart disease in childhood, and it is not till the age of work and strain, from the age of fifteen upwards, that aortic disease in any form, and mitral constriction, begin to appear as common affections. There is, however, a class of cases of no inconsiderable magnitude which admits of doubt as to the exact nature of the disease. I allude to cases in which the first sound is thick, perhaps with an occasional short systolic bruit, generally with a too forcible and a diffused impulse, and some irregularity. These cases no doubt may be construed as examples of slight thickening of the valve of congenital origin, and as being the cases which will eventually become contracted mitrals of pronounced form. I confess I do not think so, because I have had now many opportunities of watching such cases for many months, and the physical signs alter and improve so much under persistent hæmatinic treatment that I believe most of them are really due to abnormal muscular action, and not to the thickening of the valve. Such children are generally pale, and often excitable and nervous, and arsenic and iron by improving these symptoms moderate the cardiac ones also. At the same time I believe that if these cardiac symptoms persist for any length of time they are of themselves efficient to originate endocardial thickening—chronic inflammation of the endocardium, hypertrophy of the endocardium, or whatever we may choose to call it—and thus to lead to mitral constriction in the course of years, as surely as chronic urethritis leads to stricture, and this without any necessity for invoking the aid of congenital malformations. This is not merely an interesting speculation upon the etiology of valvular disease of the heart, it is a hypothesis which if true has practical bearings, the importance of which it would be difficult to over-estimate.

Chorea is another evidence of rheumatic strain. I should have felt some diffidence in adducing any evidence on this point as I had thought that all of us were quite sufficiently assured of it. But since no less careful an observer than Dr. Sturges, of the Ormond Street Hospital, disputes it, perhaps the question may still be considered an open one. Dr. Sturges disputes the rheumatic origin of chorea on this ground, that only a small proportion of choreic children have had acute rheumatism, and to the criticism that any attempt to settle the affinities of chorea is valueless which does not take into account such family proclivities as exist, he replies that we do not as yet know the proper share of rheumatism that is to be accorded as an average to each family.

It may be admitted that there are many difficulties in the way of accurately tracing such a disease as rheumatism in families, but these are by no means insuperable, and I believe as much can be said of rheumatism as of other strains, that some families are markedly rheumatic, while in others no history of rheumatism can be obtained; and it seems to me sufficiently obvious that if there is any truth at all in the doctrine of heredity this must be so, unless, as is contended by some, rheumatism is a disease which is easily acquired.

From some observations I have made upon this point, however, I believe that taking 100 patients consecutively, without any selection, about 30 per cent. will be found to give some history of rheumatism in close relatives—using the term rheumatism in a comprehensive way—and if we restrict it to illness which has confined the patient to bed with pain and swelling in the joint for a period of weeks, it becomes of course much smaller. I see no ground therefore for supposing that acute rheumatism is readily acquired. If it were it ought to appear more indiscriminately than it appears to do. Having said this I may now add that a careful inquiry into family history seems to me to show incontestably that acute rheumatism and chorea are so frequently found in the same family as to prove a relationship to each other.

The summary of the facts at my disposal is as follows the notes of the cases are given in an appendix.

I have 81 cases, 57 of them in girls, 24 in boys.

Auto-rheumatic only	9
Auto-rheumatic, with family history	10
Family history only	35
Gouty family history	2
Not stated	4
No history of rheumatism known	21
					<hr/> 81

It may further be stated that twenty-three of the cases had permanent mitral disease. In three others there was questionable disease, two had aortic disease, and one both aortic and mitral disease. Two of the mitral cases had a præsystolic bruit. A history of fright is given in five cases, but in all of them there was either a history of previous rheumatic fever in the patient or evidence of family taint of rheumatism.

Is it only a coincidence that two thirds of the cases have a rheumatic taint of some sort, viz. exactly the same proportion as was found in the cases of acute rheumatism and rheumatic heart disease previously given, and as Dr. Duckworth obtained for the rheumatic origin of constricted mitral?

The position I take with regard to chorea then is this, that in two thirds of the cases it has close rheumatic relations, but I do not think it is always rheumatic by any means; the remaining third of the cases is a good margin for the working of other causes, of which probably mental shock may count as one; various forms of aberrant nerve functions in the progenitors, such as epilepsy, neuralgia, as others; gout as another; and probably minor traits, which in the course of descent become gradually intensified till they culminate in chorea, but which on looking back present no sufficiently decided type to justify the allotment of a particular name.

Nightmare, or night terrors, stands next upon my list as a condition which is liable to occur in rheumatic children. This is a very common affection, and naturally enough I have not taken such careful notes of these cases as I have done of heart disease, chorea, &c.; but I have notes of 37 cases, 21 boys and 16 girls—17 of them, 12 boys and 5 girls, had a family history of rheumatism, 5 others came of nervous or neuralgic stock, and in 8 the point was not inquired into. In 7 there was no history of rheumatism of any kind. I may also add that only 3 of the 37 are noted as rachitic.

Headache.—Obstinate headache in children is frequently found in rheumatic families. I have notes of 83 such cases. Twenty-three of them were of rheumatic stock, 5 of epileptic, 5 showed no abnormal taint. With some of these there was associated an intractable anæmia.

Anæmia.—Acute rheumatism in the adult has been shown to be associated with a deficiency in the corpuscular elements of the blood, and there is a large group of cases of anæmia in childhood associated not with rheumatism itself but with the rheumatic strain. I am inclined to think, further, that it is more common in girls than in boys, and in those of dark rather than those of light complexion. But this is opposed to what is generally taught concerning acute rheumatism.

Neuro-muscular derangements form another important group of cases found in those of rheumatic strain. Irregularities of muscular action, other than chorea, muscular spasm, &c., of which I may mention some cases of cervical opisthotonos, of torticollis, tetany, muscular tremors, œsophagismus and stammering, incontinence of urine, felicitously termed by Sir James Paget the stammering bladder, also constitute a liability attached to rheumatism, and last, but by no means least, I would allude to a state of irregularity of bowels, which seems to me to be of very much more interest than such a common place incident might at first sight appear. There are a number of children seen in the out-patient room of whom the tale is that they have frequent attacks of severe abdominal pain, the motions being loose and quickly succeeding a meal. Sometimes it is said of such children that as fast as they eat the bowels act. Now these are symptoms which are attached to *tabes mesenterica*, and at first I was inclined to believe them, when persistent, to convey this indication. Of late, however, I have come, I think, to recognise that some of them are of rheumatic origin or, to put it more generally, are associated with a rheumatic strain, and that the symptoms are due to an abdominal neurosis. It is a condition which occasionally persists to adult age, and very inconvenient the affection is. I have an out-patient who comes to see me occasionally, very nervous he calls himself, but his chief trouble is that his bowels act upon the slightest excitement.

We are all familiar with the occasional manifestation of this form of nervous discharge, under the influence of great fear, and so on. But my point is this, that what occasionally occurs as an acute intestinal convulsion also presents itself as a chronic convulsive disease, an abdominal epilepsy or paralysis agitans may we call it, and as such requires recognition and treatment.

I have already alluded to the interesting reflections which arise by bringing into juxtaposition the acute joint affection of acute rheumatism and acute nerve change, and the chronic joint affection of chronic rheumatism and chronic nervous change; it does not diminish this interest to find that these abdominal affections allow of a similar grouping, and that while ataxie locomotrice has its well known crises gastriques, the rheumatic strain has also an abdominal spasm or convulsion, such as I have described.

Well, then, a number of children are brought of whom the mothers only complaint is, that they are "*so nervous*." Now, this sometimes means that a child is choreic, but more often no more than that it is unnaturally timid. Perhaps it will scream if left in bed in the dark, or if asked to go upstairs by itself in the dark. Sometimes it is that at games the child becomes so morbidly excited that it is quite exhausted and ill afterwards. All these things point to excessive nerve discharge for abnormally slight stimuli. The symptoms are well comprised, I think, in the mother's term "*nervous*;" and such conditions I say are prone to occur in children whose parents or relatives suffer from rheumatism.

I will allude to the *cutaneous affections* of the rheumatic child, only to say that erythema nodosum and psoriasis are not uncommonly evidence of the parentage of the child. Thus in twenty-nine cases of erythema nodosum, nineteen were rheumatic, five were not, and five others were not interrogated upon the subject. Thus here again we have cutaneous affections in the course of rheumatism which either by their course or associations suggest their nervous origin.

And now let us summarise and see to what our facts lead us.

I have endeavoured to show that parents who have had rheumatic fever transmit to their offspring a something, a con-

stitution, which tends to show itself in various ways; sometimes by acute rheumatism; sometimes by the slow production of endocardial thickening and valvular disease; sometimes by bad headaches; sometimes by obstinate anæmia; sometimes by irregularities of muscular action, such as chorea, occasionally by epilepsy, by abdominal convulsion, or perhaps it may be as well to adopt Charcot's term as a general one, and call all such crises *gastriques*, sometimes by a more general but persistent low tone of nervous system, such as may happen temporarily to any one when below par, but is then speedily recovered from.

As I look over my notes, and one by one the varied affections which occur in rheumatic families are filled in, the picture takes the outline, hazy as it should be, but still an outline of what has been called of late years by Dr. Edward Liveing a nerve storm; and when we further call to mind that cases of acute rheumatism are on record which have been intimately associated at their onset with mental shock, the neural phenomenon of hyperpyrexia, and so on, there is no small ground for the view that rheumatism is engendered by, or engenders, a state of nervous instability. It is nervous in the multiplicity of its forms; it is nervous in its associations; it is nervous in its points of attack; it is nervous in its methods of attack, in its course and progress; and it is nervous in its treatment. And although the most striking phenomenon of acute rheumatism, the acute synovitis has no very evident connection with nervous instability; although we can as yet say but little, say nothing, of the nature of the process, I have already said in anticipation we know as a fact that other acute inflammatory processes are intimately associated with lessening and destruction of nervous influence; and little as we know of the nature of the bond which exists between them and the destructive joint affections, acute and chronic, which occur in such diseases as myelitis and ataxie locomotrice, the fact of their association is among the possessions of pathology.

The neurotic origin of gout has always had numerous supporters. Surely, if so, rheumatism may have also; and even osteo-arthritis has many points in its history and associations which favour the opinion that it too is of neurotic origin.

I am even inclined to go further, and to say that the study of these diatheses suggests that gout, rheumatism, and osteo-

arthritis, although, no doubt, distinct in the individual, are all first cousins to each other, and that they are very possibly modifications or varieties of some common ancestral less specialised type, which in the process of evolution either of the disease or the family has given rise to all.

It is not, however, with such a speculative subject as this that I would end this paper. There are many points of the greatest practical moment involved in the question I have attempted all too crudely to discuss. I must content myself with touching only upon one, and that one I may call the beginnings of heart disease.

I have given facts to show that two thirds of all the cases of heart disease in children are of rheumatic origin. I have further given evidence for the belief that rheumatism is one of the family of neuroses; and if so then heart disease may own in many cases a nervous origin. Let us go to work with this in view and what do we find? Why, that there is clinical evidence of disturbed cardiac action in all sorts of cases of the so-called functional nervous disease. Not evidence of pronounced heart disease, were it so there would be less need to call attention to it, the fact would be too patent, but evidence of muscular irregularity; displacement of the impulse; abnormal quality of sounds; occasional and temporary mitral bruits, and so forth, which *must* indicate increased wear and tear to the organ concerned. Let such a condition go on, as it does often, if not treated, for months, and is it not likely, to say the least, that thickening of the valves will be slowly engendered, which when started is difficult to arrest? But on the alert to recognise the slight traits of habit and of manner—the pallor, the neuralgia, &c., which betoken what I have ventured to call a diathesis, and its attendant risks, those who have the medical charge of families are able to arrest by timely treatment these earlier functional diseases of the heart and avert the production of permanent valvular disease. If any other incentive to vigilance were necessary I would say that the careful observation and record of series of such cases would form a most valuable contribution to the etiology of heart disease, which, as all must know, is a subject than which hardly any other is more vital from the frequency of its occurrence, or more interesting from the many problems it offers for solution to the intelligent mind.

APPENDIX.

I have thought it well to add here a short note of all the cases of acute rheumatism, heart disease, and chorea, upon which my conclusions are based. The prefix attached to each case indicates the relation to previous rheumatism: G = good family history; M = moderate family history; I = indifferent family history; R = rheumatic fever in the patient only; N = absence of history. Imperfect notes are indicated by —.

1. ACUTE RHEUMATISM.

I. Girl, aged 6. Mother rheumatic in head, shoulders and wrists. Heart normal.

M. Boy, aged 8. Maternal grandfather has rheumatic gout. Heart normal.

G. Girl, aged 9. Father has had rheumatic fever. Heart sounds thick; impulse external to nipple.

G. Boy, aged 9. Mother has had rheumatic fever. Heart normal.

— Girl, aged 3½. Family history not stated. Heart normal.

— Boy, aged 12. Family history not stated. Heart normal.

G. Girl, aged 4. Father died with rheumatic fever and heart disease. Rheumatic fever came on after scarlatina in patient. Heart (?).

R. Girl, aged 2½. Sister has night terrors. Baby brother convulsion. This child has rheumatic fever after scarlatina.

G. Boy, aged 7. Mother has had rheumatic fever. First sound thick.

G. Girl, aged 8. Mother has had rheumatic fever three times. Heart's action very rapid; no bruit or displacement.

G. Boy, aged 3½. Sister had contracted mitral under me; since dead. Heart normal.

M. Boy, aged 9. Paternal grandfather has gout. Father has "rheumatism," but never laid up. Heart normal.

N. Boy, aged 3½. Family history none. First sound prolonged.

G. Girl, aged 10. Mother has had rheumatic fever. Child's second attack. Heart hypertrophied and mitral incompetent.

G. Girl, aged 3 months. Mother had rheumatic fever. Heart normal.

N. Boy, aged 8. No family history of rheumatism. Heart, systolic apex bruit.

G. Boy, aged 6. Mother has had rheumatic fever. Heart normal.

I. Girl, aged 5. Father subject to "rheumatism," never laid up. Disease came on after scarlatina (?).

M. Girl, aged 4. Two maternal uncles have had rheumatic fever. Heart normal.

I. Girl, aged 12. Mother has had rheumatism. Heart normal.

G. Boy, aged 8. Mother has had rheumatic fever. Heart's action forcible.

M. Boy, aged 2½. Maternal grandmother has had rheumatic fever. Heart normal.

G. Boy, aged 10. Father has had rheumatic fever. Heart normal.

M. Boy, aged 4. Paternal uncle and maternal uncle have each had rheumatic fever. Heart's impulse diffused, with mitral systolic bruit.

M. Girl, aged 8. Mother has been subject to rheumatics since childhood. Heart normal.

N. Girl, aged 6. No family history. Heart normal.

— Boy, aged 8. Family history not stated. Second attack associated with purpura and mitral regurgitation.

G. Boy, aged 2. Mother has had rheumatic fever. Heart normal.

N. Girl, aged 10. No family history. Heart doubtful; sounds muffled and thick.

G. Girl, aged 6. Father has had rheumatic fever. Heart normal.

G. Girl, aged 4. Paternal grandfather and maternal aunt have had rheumatic fever. Mother has had rheumatics, but never laid up, and father also. Her own sister has pains in her limbs, and a double murmur.

—, Girl, aged 8. Family history not stated. Heart normal.

R. Girl, aged 6. No family history. Acute rheumatism after fright? Heart normal.

G. Girl, aged 11. Her mother died of heart disease, and her brother has had rheumatic fever. First sound thick.

G. Girl, aged 6. Mother had *chorea* when pregnant with this child. This child has had *chorea*. Short, soft systolic apex bruit.

N. Girl, aged 8. No family history. Heart normal.

—, Boy, aged 7½. Family history not stated. Heart normal.

R. Girl, aged 5. Family history not stated. Rheumatic fever after scarlatina.

G. Girl, aged 13. Father was laid up a long time with rheumatism. Heart normal.

I. Boy, aged 12. Mother has had rheumatism. Heart normal.

N. Boy, aged 12½. No family history. Heart normal.

M. Girl, aged 4. Paternal grandfather long laid up with rheumatism. Heart normal.

N. Girl, aged 7. No family history. Heart normal.

G. Girl, aged 11. Mother had rheumatic fever. This child has mitral disease and pericarditis.

2. RHEUMATIC HEART DISEASE.

M. Boy, aged 8. Mother laid up once for a week with rheumatism during a confinement. Patient laid up with rheumatism a year ago for two or three weeks; now has frequent pain in all his joints. Heart's impulse diffused all round the nipple, with systolic thrill in fifth and sixth spaces; action very rapid; first sound accompanied by short, loud systolic bruit, audible in axilla and back.

N. Girl, aged 9. Subject to rheumatic pains, but never laid up. No family history of rheumatism or gout. Dark aspect. Cardiac impulse in fifth space outside nipple. Heart's action, irregular; systolic bruit at fourth interspace to left of sternum. Slight systolic roughness over the aortic valves.

I. Girl, aged 6. Complains much of pains. Heart irregular. Father has rheumatics.

M. Girl, aged 8. Complains of cough. Heart's action irre-

gular, and occasionally two short beats followed by a short pause; no bruit; second sound very accentuated over the pulmonary cartilage; impulse normal in position, but heaving and forcible. Mother rheumatic, and father crippled by it so that he walks on crutches. Sister has it in feet.

R. Girl, aged 3½. Had acute rheumatism seven weeks before her attendance. Præcordial dulness increased; impulse low down and extending outwards into mid-axillary line. Slight thrill in ensiform cartilage region. Loud systolic bruit extending into axilla and back. No family history.

I. Girl, aged 8. Pain in left side for twelve months. Præcordial dulness increased; impulse forcible, vertically below nipple in fifth space; no bruit. Maternal aunt has rheumatism.

G. Girl, aged 11. Had rheumatic pains, but never laid up. Chest prominent in front. Heart's impulse forcible and external to the nipple. No bruit, but a thumping and vibrating action of heart with irregularity. Father subject to pains in the limbs, and mother laid up eighteen months with "rheumatic fever and gout."

I. Girl, aged 11. Complaint of headache. Loud mitral systolic bruit. Said never to have had rheumatism herself, but father has rheumatism in his legs.

M. Boy, aged 9. Has had three attacks of rheumatic fever. Heart's apex in sixth interspace an inch outside the nipple over an area of one and a half by two inches. Systolic murmur at the apex, in axilla, and between the scapulæ. Maternal grandfather subject to rheumatism; paternal uncle has had rheumatic fever.

G. Girl, aged 12. Extreme cyanosis. Quite well till five years ago, when she had scarlatina; since then she has always been weakly, with pain in her chest, and for two or three years she has been getting blue. Heart: apex beat diffused, but most marked at normal spot, slight thrill at apex. Systolic apex bruit fading in intensity rather quickly towards the axilla. Loud systolic bruit over the aortic valves, third costal cartilage, mid-sternum; over mid-sternum, third rib level, is a loud, long, whiffing bruit, much louder than that over the aortic region, and heard all over the left side. No venous pulsation in neck. Her father has had rheumatic fever, and was laid up a long time. Diagnosis: (?) Tricuspid bruit, with regurgitant,

mitral, or aortic systolic, or congenital absence of part of the septum.

G. Boy, aged 6½. Rheumatic fever two months ago. Diffused impulse, with slight thrill; apex in third space; a local systolic bruit at base. Father has had rheumatic fever.

G. Girl, aged 11. Subject to rheumatic pains in her joints. Impulse diffused; apex beat within and without the nipple; first sound a prolonged thump; action irregular. Father very subject to rheumatism, and several of his family have had heart disease.

G. Boy, aged 10. Gradual wasting for twelve months, with much visible pulsation. Pale. Heart dulness large, impulse diffused; sounds lumpy; second sound loud. Mother had rheumatic fever while nursing child; ill five months.

G. Boy, aged 11. Frequently has pain all over him, and goes off into a faint. Heart's impulse diffused all round the nipple; there is no bruit, but the first sound is particularly thick and muffled at the apex. Impulse heaving; action rather irregular; second sound loud. Father has had rheumatic fever.

I. Girl, aged 10. Six weeks ago had erythema nodosum. Complaints of palpitation. Is overgrown. Heart's impulse diffused in fifth space to half inch external to nipple. Action irregular; sounds thick; no bruit. One or two of family have "rheumatism."

G. Girl, aged 9. Headache six weeks. Heart's impulse forcible, diffused, and long, with an occasional short bruit at apex. Mother has been laid up in bed with rheumatism for three months.

M. Girl, aged 8. Cough complained of. Systolic bruit at apex. Maternal grandmother had rheumatic fever.

G. Girl, aged 11. Very pale. Impulse very diffused in fourth and fifth spaces outside the nipple. Short systolic bruit with loud ringing second sound. (?) Hypertrophy with dilatation and adherent pericardium. Mother has had rheumatic fever and has heart disease.

I. Girl, aged 6. Has cough. Reduplicated second sound at apex, both on auscultation and to touch. No thrill and no præ-systolic bruit. Maternal grandfather and great aunt subject to rheumatism.

I. Boy, aged 4. Poorly for last six months and wasting. Dark aspect; anæmic. Impulse of heart in fifth space vertically below the nipple. Loud systolic bruit all over the front of heart, but loudest at the base; audible behind over the vertebra prominens. Apex bruit (?). No rheumatism in family but gout on father's side.

R. Girl, aged 11. Rheumatic pains in joints with swelling. Heart's impulse in fifth space under nipple. Loud systolic murmur audible posteriorly.

R. Boy, aged 7. Systolic bruit at apex with occasional double pericardial friction sound and pleurisy (?). Impulse diffused outside the nipple. No family history. This boy had rheumatic fever two years and a half ago; was ill a long time.

G. Boy, aged 6. Short breath for a few days. Heart's impulse forcible, external to the nipple and below sixth rib. Præcordial dulness large. Systolic apex bruit in axilla and behind; systolic bruit also; (?) diastolic over the mid-sternum. The boy was in the hospital three months before for rheumatic fever. Father has been laid up with rheumatic fever.

G. Girl, aged 3½. Cough for about a fortnight. Bruit; harsh, short and systolic from mid-sternum upwards to left. Mother has been laid up with rheumatic fever for three months.

G. Boy, aged 4½. Has had a bad cough for some months. Heart's impulse diffused, external to nipple. Action irregular, first sound at apex long. Mother has had rheumatic fever.

R. Girl, aged 11. Pain in side and wasting. Loud local systolic bruit, with a questionable systolic at the impulse; loudly systolic behind. Has had rheumatism several times. No family history of rheumatism.

R. Girl, aged 8. Has always been delicate; rheumatic pains about her for a month. Increase of epigastric pulsation, and also of impulse at nipple. Area of dulness not increased. Action regular. No bruit. Mother has had rheumatic fever.

I. Boy, aged 8. Cough. Pain in the heart. Wasting. Impulse vertically below the nipple. Systolic bruit at apex and away towards the ensiform cartilage. Action regular. No history of either rheumatism or gout in parents, but grandfather had gout and died of it.

I. Girl, aged 6. Wasting, "goes so yellow." Heart's

impulse diffused. Short systolic bruit after the first sound and in addition to it. Paternal grandfather had gout and all paternal uncles. No rheumatism known.

I. Girl, aged 10½. Rheumatic pains. Heart's impulse diffused to left of nipple; first sound prolonged and blowing. She has had rheumatic fever. Her father has been subject to rheumatism, but has never had "the fever." A subsequent note to this case records the presence of presystolic bruit and thrill.

G. Boy, aged 11. Mother had rheumatic fever when she was six. Heart's impulse is in the fifth interspace vertically below the nipple. Much too forcible and occasionally murmurous.

G. Girl, aged 11. Father had rheumatic fever in 1850. The patient's elder brother has had rheumatic fever. She has often complained of "pains." She is a dark thin girl with violent palpitation going on. The heart's impulse is diffused in the fourth, fifth, and sixth spaces, both in and outside the nipple line. The sounds are thick but there is no bruit. (?) Adherent pericardium.

R. Girl, aged 5. Family history not mentioned. The child had acute rheumatism last year. The heart is large; there is a loud systolic bruit in the axilla and back.

R. Girl, aged 12. Family history not mentioned. Has had acute rheumatism twice. Heart large; loud systolic bruit heard all over the chest and back.

G. Girl, aged 4 months. Mother has had rheumatic fever seven times. The child has been blue from her birth. Heart's action quiet. Action regular. No thrill. There is a short, harsh systolic bruit, loudest between the nipple and sternum over the fourth rib, but audible at the nipple and at the sternum. Second sound accentuated at the base.

G. Boy, aged 6½. Father had rheumatic fever twice. Mother had rheumatics in her knees for five weeks. Heart's action quick; impulse forcible and vertically below nipple. First sound long but no bruit.

I. Girl, aged 9. Mother "rheumatic" but never laid up. She has suffered from abdominal pains at times, and has had pains all over her for a week. Much pallor. Heart's impulse diffused. Pericardial rub over all the præcordial region, and a localised systolic apex bruit.

R. Boy, aged 6. Family history not mentioned. Quite well till two months ago, when he had pains in his back and limbs. Latterly he has had swelling of the legs. Much pallor. Præcordial dulness extensive. Impulse diffused. Endocardial fremitus communicated to the surface. Loud musical systolic bruit at the apex in axilla and back. Second sound reduplicated over pulmonary artery. No aortic or tricuspid bruit.

I. Girl, aged 8. Mother has had rheumatics but nothing of importance. Patient has never had rheumatism. Always ailing. Systolic bruit over third left costal cartilage and sternum at same level. Second sound very accentuated. Impulse felt far into the axilla in third, fourth, and fifth spaces. A systolic bruit heard inside the nipple. Five months later the bruit remained the same. She came complaining of bruising so readily and with several bruises on the shins and knees.

I. Girl, aged 6. Father has had lumbago badly. A cloud of albumen in the urine. Much pallor. An apex systolic bruit. Three months later the impulse of heart forcible; no bruit. Urine normal. Four months later she was quite well.

R. Boy, aged 16. No family history of rheumatism. Has had rheumatic fever three times. Heart's impulse diffused in fourth and fifth spaces from inside to outside the nipple; double second sound at the apex; slight systolic bruit; systolic retraction in fourth space.

G. Boy, aged 10. Mother has had rheumatic fever, ill for six weeks. Impulse of heart forcible and diffused; systolic retraction below the apex; no bruit.

R. Boy, aged 12. No rheumatic family history. Had rheumatic fever five years ago, and since then has been subject to rheumatism. Diffused impulse outside the nipple in the fifth space. A loud systolic bruit at apex audible at the back, with thick double second sound at the apex, accentuated at the base.

G. Boy, aged 16. Mother died of heart disease. He has had rheumatic fever in Guy's. Impulse forcible in fifth space vertically below the nipple. Præcordial dulness increased. First sound thick with an occasional bruit. Enlarged heart. Adherent pericardium. Thickened valves without much incompetence.

G. Girl, aged 8. Father has been laid up with "rheumatics" and mother has had it in her knees, being laid up eight weeks

and twelve weeks respectively. *Præcordia* bulging. Heart's impulse diffused far outside the nipple. Loud systolic bruit all over front and back of chest.

R. Girl, aged 13. Family history not recorded. She has had rheumatic fever, and has now advanced mitral disease.

G. Boy, aged 10. Mother has had rheumatic fever. Ailing for two months after a fright. Pallor. *Præcordia* bulging. Impulse diffused. Diastolic thrill. Distinct whiffing systolic bruit midway between nipple and ensiform cartilage. Much accentuated second sound at base.

G. Girl, aged 16. Mother had rheumatic fever. A few weeks back she had swelling of knees and wrist. Heart's action diffused. Slight systolic thrill. Loud systolic bruit all round the nipple, in axilla and back. Twelve months later this note is made:—Impulse diffused in fifth space far into the axilla. Heart's action cantering, with occasional systolic bruit. Thick sounds in axilla. The sounds give one the impression that the mitral valve is thick and narrow, but only moderately so. The mitral has probably contracted between the two attendances.

G. Boy, aged 15. Mother died of heart disease. She had rheumatic fever four or five times. Has had heart disease four years, and been in other hospitals. *Præcordia* bulging. Much increase of dulness. A loud systolic bruit all over the *præcordia*. Heart evidently much enlarged.

G. Boy, aged 13. Two brothers and two sisters have been under me for heart disease or rheumatism. Maternal grandmother suffered from rheumatic gout. Has had rheumatic fever twice. Heart's action heaving. Impulse in fifth inter-space inside nipple. Loud systolic apex bruit; somewhat local in its point of greatest intensity; not heard in the back.

G. Boy, aged 10. Same family as previous case. Said to have had rheumatic fever after scarlatina. Impulse external to nipple. Loud regurgitant mitral bruit, heard all over back and front.

G. Girl, aged 8. Same family as the two preceding cases. Rheumatism four years ago, and since then three other attacks. Heart's impulse in the sixth space, in and outside the nipple line. Systolic apex bruit, with double second sound, heard from sternum to nipple and into the back.

G. Girl, aged 7. Same family as the previous three cases. Has had rheumatism (?) after scarlatina. Heart's action irregular; first sound rough, second persistently accentuated and sometimes double. No thrill, but impulse is over forcible.

G. Boy, aged 4. Same family as preceding four cases. Has had rheumatics all the winter; not kept his bed, but has been dressed and put in a chair. The heart is normal.

G. Boy, aged 6. Mother has had rheumatic fever. Heart, thick first sound at apex.

I. Girl, aged 5. Paternal grandmother suffered much from rheumatics. Heart's action cantering; sounds muffled; action rapid; apex diffused; short, rough, diastolic bruit. Swelling of knees and feet now present.

I. Boy, aged 8. Father has rheumatics in back and ankles. Came first with loud systolic apex bruit, and subsequently developed a pericardial rub.

I. Girl, aged 9. Maternal grandfather rheumatic. Has pains. Heart's impulse thumping and diffused outside the nipple. Slight systolic bruit in fourth space, midway between the nipple and sternum.

G. Boy, aged 6. Father has had rheumatic fever twice. He has had rheumatic pains about him. Heart's impulse diffused in fifth space outside the nipple. First sound thick, and an occasional short whiff.

M. Girl, aged 11. Father and paternal uncle suffer much from rheumatic gout. Mother has fits. Heart's impulse forcible. First sound thick and long.

G. Girl, aged 5. Father has had rheumatism and was laid up for some months. A loud systolic bruit at apex. Impulse forcible below and external to nipple.

?. Girl, aged 12. Rheumatic family history probable. Heart's impulse forcible in fifth space. External to nipple, first sound peculiarly loud and high pitched, followed by a hoarse systolic bruit, loud in axilla and back.

I. Girl, aged 7. Father subject to rheumatism, never laid up. Ailing since the scarlatina, six months ago. Heart's impulse diffused in and outside nipple; loud systolic bruit all over heart's area, with accentuated second sound. Bruit very loud in the back.

G. Girl, aged 17. Maternal grandfather had rheumatic fever

badly three times. Mother has rheumatism occasionally. Girl had rheumatic fever four years ago. Heart's impulse forcible and diffused; loud systolic apex bruit audible in the back.

G. Girl, aged 9. Brother had rheumatic fever. Marked anæmia. Impulse forcible, external to nipple. First sound thick and distant, second accentuated at base; no bruit.

G. Girl, aged 7. Mother has had rheumatic fever. Child had "growing pains." Heart impulse very forcible in fifth space, half inch external to nipple. Slight musical systolic bruit round apex. Doubtful in the back.

M. Girl, aged 5. Paternal grandfather has rheumatic gout. Heart impulse diffused outside nipple. First sound thick and musical, second occasionally double at the apex.

M. Girl, aged 5½. Mother had rheumatics, but never laid up with it. Child has frequently cried with pains in her limbs. Heart's action very rapid and cantering. Systolic thrill; diffused impulse; apex far outside nipple; loud systolic bruit.

R. Girl, aged 9. Family history not stated. Has had rheumatic fever. Long and loud systolic bruit at the apex. The bruit is a little musical, and preceded by a very noisy first sound. Impulse in sixth space, one and a half inches external to the nipple; systolic thrill; bruit loud at the angle of scapula.

R. Girl, aged 7. Has had rheumatic fever three times. Family history omitted. Loud systolic bruit with very loud ringing first sound; impulse in fifth space vertically below the nipple.

G. Girl, aged 11. Father has had rheumatic fever. Heart's action tumultuous and irregular; no bruit; impulse forcible.

G. Girl, aged 9. Brother under my care for scarlatinal rheumatism. This child under me four years ago for "heart disease." Notes of case lost. Impulse now diffused in fifth space inside the nipple, and the first sound is thick with a little reduplication of second sound. Nothing else to be noted.

G. Boy, aged 8. Father had rheumatic fever four times. This boy had it when five. Frequent palpitations. Heart's action forcible in fifth space. Action regular. Sounds very thick and distant. (?) Adherent pericardium.

G. Girl. Mother has had rheumatic fever. Child has had it before. Loud mitral bruit with extensive pericarditis.

G. Girl, aged 9. Sister died of heart disease. Heart's action quick and forcible in fifth space beneath the nipple.

G. Girl, aged 7. Mother has had acute rheumatism twice. One brother has also had it while under my care. Heart's impulse forcible. A loud hoarse bruit consisting of systolic and præ systolic portions. Heard loudly in axilla and back. Slight thrill at apex.

R. Girl, aged 11½. No family history of rheumatism to be obtained. She has had rheumatic fever. Loud apex systolic bruit heard in axilla and back.

G. Girl, aged 11. A brother or sister died of heart disease. No other evidence of rheumatic taint. Heart's action irregular. Impulse in normal position but forcible and grating. No distinct bruit.

R. Girl, aged 10. No family history of rheumatism. Had rheumatic fever once for five weeks. Rheumatism frequently. Heart's impulse forcible, far external to nipple. Slight thrill. Loud systolic musical bruit back and front.

G. Girl, aged 4½. Father had rheumatic fever eleven years ago. She has had it. Advanced mitral disease with acute pericarditis and dilatation.

R. Girl, aged 6. No rheumatic history in family. Her brother aged eleven months was brought with a curious neuromuscular fault in the action of fauces and œsophagus. She has had rheumatic fever. Diffused impulse, no bruit.

M. Girl, aged 12. Maternal grandmother had rheumatics badly, and rheumatic fever. This child has had pains in her bones and chorea. Heart's impulse far outside the nipple. Slight thrill. Systolic apex bruit.

M. Boy, aged 8. Family history of heart disease. None of rheumatism. Systolic apex bruit.

R. Girl, aged 6. Family history not mentioned. Has had rheumatic fever and chorea, also pericarditis. Loud systolic bruit at the apex and in back.

G. Boy, aged 5. Father has had rheumatic fever three times. The child not known to have had it. Systolic bruit at the apex.

G. Girl, aged 8. Sister died in hospital with heart disease. Extensive præcordial dulness, and diffused impulse. Very thick and accentuated second sound. (?) Adherent pericardium with thick valves.

R. Boy, aged 6. No rheumatic family history. He has had it. Loud mitral bruit with ascites and anasarca. He was relieved by paracentesis, &c., and still goes along pretty well three years after.

I. Boy, aged 4. Rheumatism for four months. Pallor. Loud, long systolic bruit round the impulse. Father has rheumatic pains but has never been laid up with the fever. Mother healthy. Patient has not had chorea.

M. Girl, aged 6. Double murmur at apex. Systolic at back. Impulse vertically below the nipple. Maternal aunt has had rheumatic fever. Mother has had rheumatism, but was never laid up with it. Father has also had it, especially in changing weather, but is not laid up with it.

G. Boy, aged 14. Brother had rheumatic fever. Patient had rheumatism for three months four years ago. Heart's impulse in fifth space inside the nipple. Systolic bruit following a thick first sound.

R. Boy, aged 11½. Family history unknown. Rheumatism and swelling of joints three weeks. Heart's action quick; impulse diffused and external to the nipple. First sound soft and thick.

R. Girl, aged 10. One other child suffers from headache. Rheumatic fever two years ago. Loud mitral systolic bruit; a second systolic bruit of musical quality at ensiform cartilage; (?) tricuspid.

R. Girl, aged 16. Rheumatic fever four years ago. Impulse in sixth space beneath the nipple. To and fro aortic bruit, and at apex the first sound is long, loud and muffled, with much the character found in some cases of contracted mitral.

G. Girl, aged 16. Mother has had rheumatic fever. Patient has had two attacks of rheumatism. There is a long thrill, chiefly diastolic, but also systolic. Impulse forcible in fifth space half an inch external to the nipple. Bruit a typical præ-systolic followed by a diastolic roll.

? Girl, aged 10. History of rheumatism doubtful on father's side. She has frequently been laid up by aching in her limbs. Very forcible thrilling impulse diffused in the fifth space external to nipple; first sound very noisy, and very short, distant systolic bruit following it.

G. Girl, aged 14. Mother has had rheumatic fever. Child

has fluid in one knee and enlargement of both wrists. Heart's action rapid; first sound thick, and distant whiffing prolongation of first sound in the left vertebral groove.

3. CHOREA.

R. Girl, aged 9. Family history free from rheumatism. She has had rheumatic fever. Heart normal.

G. Girl, aged 6. Paternal aunt had rheumatism at twenty-eight. Mother has had rheumatic fever, and maternal aunt had rheumatic fever several times. Heart, mitral systolic; (?) præsysstolic.

N. Girl, aged 13. No history of rheumatism. Systolic apex bruit.

G. Girl, aged 4½. Father has had rheumatic fever. Patient has frequently suffered with pains in her limbs. Heart normal.

N. Girl, aged 9. No known history of rheumatism.

N. Girl, aged 10. No known history of rheumatism. Heart normal.

R. Girl, aged 10. No family history of rheumatism. She had rheumatic fever two months before. Heart normal.

G. Girl, aged 19. Father has had rheumatic fever and patient also. Systolic mitral and diastolic aortic bruit.

G. Girl, aged 9. Father has been laid up with rheumatism. Heart normal.

N. Girl, aged 10. No rheumatic history. Heart normal.

N. Girl, aged 10. No rheumatic history. Apex systolic bruit.

N. Girl, aged 9½. No rheumatic history. Heart normal.

M. Girl, aged 7. Maternal grandfather has been laid up with rheumatism. Maternal aunt and uncle have had rheumatism. Heart normal.

I. Boy, aged 10. Father has had "rheumatism." Two other children have had chorea. Heart normal.

G. Girl, aged 12. Rheumatic family history. Chorea after joint pains. Systolic apex bruit and thrill.

N. Girl, aged 7. No family history of rheumatism. Second attack of chorea. Heart, loud systolic bruit and pericardial rub.

G. Girl, aged 11. Father has had rheumatic fever. Mother has had chorea. It is the fourth attack. Heart normal.

N. Girl, aged 8. No history of rheumatism. Systolic apex bruit.

M. Girl, aged 10. Rheumatic history on father's side. Heart normal.

N. Boy, aged 10. No rheumatic history. Always nervous. Heart normal.

R. Girl, aged 10. No family history. Rheumatic fever five months ago. Mitral systolic bruit.

G. Boy, aged 13. Mother has had rheumatic fever, so also has boy. Heart normal.

N. Boy, aged 9. No rheumatic history. Heart normal.

R. Girl, aged 15. Rheumatic fever one year ago. Mitral systolic bruit.

G. Boy, aged 8. Father has had rheumatism in several joints, so also has boy. Heart, no bruit.

N. Girl, aged 13. No rheumatic history. Thick first sound and occasional systolic bruit.

— Girl, aged 12. Family history not stated. First sound thick.

R. Boy, aged 10. Has had rheumatic fever four years ago. Thick first sound, and occasional short rough bruit.

G. Girl, aged 12. Mother has had rheumatic fever, and child also. Chorea attributed to a fright. Short systolic bruit at base.

G. Boy, aged 9. Mother had rheumatic fever, and died of heart disease. Systolic basic bruit.

G. Girl, aged 16. Father laid up for long in bed with rheumatism. Heart normal.

G. Girl, aged 8. Mother had rheumatic fever and child also. Heart large; systolic apex bruit.

I. Girl, aged 14. Mother has had rheumatism in the shoulder. Father had fits. First sound thick.

I. Girl, aged 16. Mother has had rheumatism. Third attack of chorea. Heart normal.

I. Girl, aged 13. Mother has had rheumatism slightly. History of fright. First sound thick and musical.

M. Girl, aged 4½. Paternal grandfather had rheumatic fever. Heart normal.

M. Girl, aged 6½. Father has been laid up in bed with rheumatism. Heart normal.

R. Girl, aged 14½. Pains in joints three weeks before chorea. Indistinct history of fright. Systolic apex bruit.

G. Girl, aged 10. Mother has had rheumatic fever. Heart normal.

G. Boy, aged 13. Mother and one of his brothers have had rheumatic fever. Heart normal.

G. Boy, aged 8. Father died of diseased heart. Chorea three months. Loud systolic apex bruit.

G. Girl, aged 5. Father has had rheumatic fever. Chorea after fright. Heart sounds coarse.

G. Boy, aged 11. Mother has articular rheumatism. No heart disease.

M. Boy, aged 10. Paternal grandfather and aunt have had rheumatism. Second attack. Heart normal.

N. Boy, aged 15. No rheumatic history. First sound thick, impulse diffused.

R. Boy, aged 5½. Rheumatic fever followed by chorea after a month. Loud apex systolic bruit.

R. Girl, aged 5½. No rheumatic family history. Has had pains in her limbs. Loud apex bruit.

R. Girl, aged 8. Chorea after pains in her joints. No family history. Heart normal.

— Girl, aged 7. Slight chorea. Heart normal. Family history not noted.

G. Boy, aged 9. Mother has had rheumatic fever. Heart normal.

N. Boy, aged 4. Mother epileptic. Heart normal.

N. Girl, aged 5. No rheumatism. Heart normal.

M. Girl, aged 4½. Paternal grandfather had rheumatic fever. Heart normal.

N. Boy, aged 5½. No rheumatic history. Heart normal.

I. Boy, aged 4. Sister had chorea, under my care. Heart normal.

G. Girl, aged 6½. Maternal grandmother had rheumatic fever, and aunt heart disease. Has had fits. Heart normal.

G. Boy, aged 11. Father had rheumatic fever. Heart normal.

I. Girl, aged 5. Father rheumatic. Heart normal.

N. Girl, aged 4. No family history. Heart thumping, with apex bruit.

— Boy, aged 5. Family history not mentioned. First sound long.

G. Girl, aged 11. Both father and mother have had rheumatic fever. Systolic apex bruit.

N. Girl, aged 11. No rheumatic history. Second attack of chorea. Previous attack two years before. Loud systolic and præ systolic bruits.

I. Boy, aged 11. Father has had rheumatics. Heart normal.

I. Girl, aged 10. Father's family rheumatic. Third attack of chorea. Heart normal.

G. Girl, aged 7. Paternal uncle rheumatic. One brother of this child had rheumatic fever and died of heart disease. A sister has had chorea. Heart normal.

— Boy, aged 9. Family history not stated. Heart normal.

G. Girl, aged 7. Father had rheumatic fever. Heart normal.

G. Boy, aged 10. Parents healthy. One brother has had rheumatic fever. Heart normal.

N. Girl, aged 6. Parents healthy. Heart normal.

N. Boy, aged 8. No rheumatic history. Heart normal.

I. Girl, aged 8. Parents healthy. First cousin, mother's side, had chorea and has heart disease. Heart normal.

G. Girl, aged 8½. Brother has had rheumatic fever, and so has she, chorea since. Heart normal.

N. Girl, aged 6. No family history. Heart normal.

G. Girl, aged 9. Mother has had rheumatism, and in bed a month. Chorea attributed to fright the night before its onset. Heart normal.

Gout. Girl, aged 11. Father has had gout for some years.

Gout. (?) Girl, aged 9. Has had erythema nodosum after scarlatina. Paternal grandfather had gout badly, and died of diseased heart.

G. Girl, aged 8. Mother has had rheumatic fever. Heart normal.

G. Girl, aged 6. Father has been laid up a month with rheumatics. Mother also has kept her bed with rheumatic gout. Heart normal.

M. Girl, aged 9. Paternal aunt has had rheumatic fever,

She has had achings in her limbs. Præsystolic apex bruit, with slight thrill.

M. Boy, aged 10. Grandmother had rheumatic fever. Child had rheumatic fever after scarlatina four years ago. Systolic apex bruit.

N. Girl, aged 7. No rheumatic history. Heart normal.

ALOPECIA AREATA.

BY P. H. PYE-SMITH, M.D.

WE may, perhaps, distinguish the following kinds of alopecia.

1. The physiological fall of hair which produces ordinary calvities is characterised by its comparative rarity among women, by its always beginning at the vertex or in the frontal region, by its spreading gradually and not by patches, by its never reaching the temples and very seldom the occiput, and, lastly, by its affecting the scalp alone. Though often accompanied by seborrhoea sicca, and less frequently by true pityriasis capitis (a branny desquamation indicative of a slight degree of superficial dermatitis), baldness also occurs without these affections, which I regard rather as concomitants than as causes. Nor is it true, as has often been asserted, that baldness depends on loss of mobility of the skin by the occipito-frontalis.

2. Alopecia as the result of febrile and other general diseases, though it often begins the process of ordinary baldness, yet is distinguished therefrom by its affecting both sexes and all ages, by the fall of hair not being confined to any region of the scalp, and by its thinning rather than completely stripping the surface affected. Moreover, secondary in origin, it also passes away of itself after convalescence, instead of being practically incurable either by nature or by art. Syphilitic baldness agrees in these characters, and its frequency apart from any other affection of the scalp, as well as its early appearance, likewise point to its ætiology as a febrile alopecia.

3. There are some cases of complete and rapid loss of hair which do not come under either of the above heads, and

which yet, I believe, cannot be classed as examples of area. They are distinguished, first, by the hair falling off almost simultaneously from the whole of the scalp, not gradually from certain regions as in ordinary baldness, nor by the confluence of separate patches as in area; secondly, by the baldness not being confined to the scalp (nor even to the scalp and beard or eyebrows as I have seen it in area), but affecting the whole of the body; thirdly, by its not following an illness. In one case of this kind the patient was a young man in perfect health, of robust habit, and wearing a full beard. Without any assignable cause he lost the whole of the hair of his body in a very short space of time. I know of two or three other cases, also in young and healthy men; and, comparing these with the most rapidly spreading cases of alopecia areata, I think they may be fairly distinguished as a separate form of baldness.

4. It is questionable whether these somewhat rare cases of alopecia universalis acquisita are pathologically to be distinguishable from the still rarer cases of congenital alopecia. In these the nails as well as the hair are affected; and, like other deficiencies of development, the condition may be hereditary. Such cases are comparable with congenital ichthyosis, especially in such marked examples as the "porcupine boy;" and still more closely with the "hairy family" of Burma, and the blue or hairless horse exhibited a few years ago in this country.

A striking series of examples of this form of baldness occurred five years ago in this hospital under Dr. Fagge, who kindly allows me to record it here. It is remarkable that the development both of hair and nails was tardy and imperfect, but not absolutely deficient.

F.¹ Born without hair or nails. Hair began to grow when he was about twenty-three years of age, and at thirty he had a full head of hair. The finger-nails also grew after puberty, but were always ill-formed, and he never had toe-nails.

¹ The notation is that of Mr. Francis Galton in his interesting work on 'Hereditary Genius.' F, G, B, S, P denote father, grandfather, brother, son, and grandson respectively, and the same letters in italic the corresponding female relationships of mother, grandmother, sister, daughter, and granddaughter. N is a brother's son, n a sister's son; U a father's brother, u a mother's brother; and, by the above rule, N a brother's, and n a sister's daughter; U a father's, and u a mother's sister.

F. Normal.

B 1. Born without nails or hair. The former appeared while teething, the latter when she was ten years old.

n. Born without hair and nails. None yet grown.

B 2. Born with hair but without nails. Died æt. seven.

B 3. Born without hair or nails. Died æt. five months.

B B 4—9. Born with normal hair and nails.

B 10. Born partly bald with ill-formed nails. Was under Dr. Owen Rees when a boy. He is now twenty-two and has a fair head of hair, but his nails are not good.

The patient herself, then nineteen years old, the eleventh and youngest of this large family, was born without hair or nails. She had in 1876 only thin lanugo over the scalp, and imperfect nails on fingers and toes.

5. There are then clear marks of distinction between these several forms of alopecia, idiopathic and secondary, and the remarkable affection known as *Alopecia areata* (Sauvages), *Area Celsi*, *Porrigio decalvans* (Willan), *Teigne pelade* (Bazin), or *Tinea decalvans*. Indeed, so peculiar is the appearance of this disease, that what is more needful to insist upon is that in spite of its well-marked characters it is a true alopecia, anatomically identical with the other forms of atrophy of the hair, though differing in its origin and course.

The first of the above titles appears to be best, since it is distinguishing and is generally accepted; or the term *area* may be used alone. Celsus did not particularly describe this variety of baldness, but applied the word "*area*" (a bare space, *locus sine ædificio*) to any form of baldness, distinguishing ἀλωπηκία and όφίαισις as varieties.¹ The *Porrigio* of Willan

¹ See Bateman's 'Practical Synopsis' (1824), p. 175, note, and Hebra's 'Hautkrankheiten,' p. 148. Neither of these authors notices that vague as was Celsus's use of both terms, *area* and *alopecia*, subsequent writers used them still more vaguely. Thus, Stephanus in his 'Vocabulorum Medicinalium Expositiones Græcæ' (1564), p. 204, quotes from the Galenical 'Defensio Medicinæ,' 'Αλωπηκία δὲ ἐστὶ μεταβολὴ τοῦ χρώματος ἐπὶ λευκότερον, δι' ἣν χρονίζουσιν αἱ τρίχες ῥιζόθεν ἀποκίπτουσιν. He also gives the following account from Oribasius of the cause of the malady:—"Alopecia vero inde nomen invenit quod vulpes, quæ ἀλωπηξ dicitur, hoc malo sæpe corripiatur. Oritur autem plerumque ex vitiosis humoribus capite contentis, sed malitia speciem ipsæ capitis color indicat: quippe albidior pituitosum, nigrior melancholicum humorem,

meant any eruption of the scalp, including true ringworm and impetigo or pustular dermatitis, and the term is now almost out of use. The appellation *Tinea* or *Teigne* depends upon the erroneous doctrine of the parasitic nature of the disease.

On this point I am entirely in accord with most modern dermatologists. I have many times sought for a fungus, and have never found the smallest evidence of its presence with one single exception. This occurred nearly fifteen years ago when I was working under the late Professor Hebra. In one of his patients suffering from area I discovered some spores and scanty mycelium close to one of the neighbouring hairs. I showed it to the professor, and he told me that he had never seen it before. He doubted whether its occurrence was more than accidental, and with my present experience I doubt it also. Hebra himself believed at one time in the statement of Gruby that the disease was parasitic, but had long changed his opinion; and I can only share in the surprise expressed by Dr. Kaposi (Hebra's 'Hautkrankheiten,' ii, p. 149, note) that the author is associated with Bazin as a supporter of the parasitic nature of area by his disciple Dr. Neumann ('Lehrbuch der Hautkrankheiten,' p. 297). It is possible that the single observation of Gruby¹ in 1843 which gave rise to the question was made upon a case of true ringworm. Neumann, who has no doubt that area is not parasitic, once, like myself, found some spores in a case of the disease, but, like myself, doubts rather the accuracy of a single observation than the accumulated testimony of his own and other's experience. In fact, M. Bazin's statements are, I believe, the only ones which rest on large experience and assert the presence of a fungus. But French dermatologists call many cases *pelade* or *teigne pelade* which in England or Germany would be regarded as true ringworm in its later stages. In M. Hardy's brilliant lecture, it is not

pallidior flavam bilem arguit." The origin of the term receives two alternative explanations in Blancard's 'Lexicon Medicum,' published at Leyden in 1702. "*Alopecia est capillorum deflavium, a lue venerea vel aliunde excitatum, ex ἀλώπηξ vulpes et πίπτω cado (!): a vulpe cujus lotum effusa dicitur reddere loca . . . vel a malo vulpi peculiari. Vocatur etiam ὀφίασις a figura . . .* Utrique huic affectui commune est, quod areatim pili decidunt, unde etiam in genere hoc malum *AREXA* vocatur."

¹ It was published by Audouin, after whom the supposed fungus was named *Microsporon Audouini*.

difficult to recognise in the swelling, irritation, and discoloration of the skin which he describes in *pelade*,¹ the characters of ringworm. I never saw at the Hospital of St. Louis an attempt to demonstrate the presence of spores in what we should call a case of area.

Apart from the microscopic evidence, the naked-eye appearances and natural history of the disease would alone disprove the parasitic hypothesis. The hairs around the affected spot are not swollen at the root nor brittle in the shaft, but are simply atrophied, like normal hairs which are ready to drop off. There is no evidence of local irritation in the hair-sac. The disease above all is not contagious, at least as we observe it in England; and it is not curable by antiparasitic treatment.²

Area is certainly more common in children and young adults than after thirty. It seems to affect both sexes equally. In most cases it probably would recover of itself, but I believe that recovery is often hastened, if not brought about, by treatment. This consists (after establishing the diagnosis) in local irritants, and, when necessary, internal corroborants. I usually begin with a lotion containing ʒiiss or ʒij of Acetum cantharidis to a pint of water. This will often cause slight erythema in children, but in adults and in many children we may increase the strength to two, three, or four drachms with advantage, letting the irritation subside whenever it goes beyond redness on to exudation. A milder and often efficient application is Lini-mentum myristicæ, which I learnt from Sir Wm. Gull when he had charge of this department. With brown hair the Unguentum iodi of the Pharmacopœia is a very useful application. Area occurs in persons of all degrees of health, complexion, and "temperament," but if the patient is pale and thin, steel is certainly useful, and I often prescribe bark or cod-liver oil, but only when indicated by some other symptom than the bald patches.

I have only seen one instance of a second attack of area

¹ 'Leçons sur les Maladies de la Peau,' 2me partie, pp. 179—184.

² The following list gives the names of the most important authorities on both sides of this question. In favour of the parasitic origin of area :—Gruby, Bazin, Hardy, and French writers generally (except Cazenave, who confounded area with vitiligo), the late Drs. Hillier and Tilbury Fox (the latter admitting it only in a very small proportion of cases). Against this opinion :—Bärensprung, Hebra, Wilson, Hutchinson, Fagge, Liveing, Duhring, and Alder Smith.

appearing after a first had been completely cured and an interval of time had elapsed.

The following is a list of the cases of alopecia areata which have come under notice in this department during the years 1878, 1879, 1880.

No.	Sex.	Age.	Duration of disease.	Present extent.	Remarks.
1	F.	20	Began at 4 or 5. Complete after measles at 11, but eyebrows and eyelashes have grown again	Complete over scalp, except slight lanugo here and there	Negative result after 6 months' treatment.
2	F.	18	Since 2 years old	Complete, including eyebrows	Treatment not attempted.
3	M.	17	Began at 14	Scalp hair grown again without treatment	Brother of No. 2.
4	F.	26	—	Patches on scalp.	
5	M.	16	—	Do.	
6	F.	40	—	Do.	
7	F.	10	—	Nearly complete over scalp.	
8	M.	23	—	Scalp and beard.	
9	M.	25	About a year	Patches on scalp.	
10	M.	24	Two months	One patch behind ear.	
11	F.	17	—	Nearly complete on scalp.	
12	M.	14	Six months.		
13	M.	15	Nine months	Patches on scalp.	
14	M.	12	Four years	Nearly complete on scalp	Dark and pale.
15	M.	19	Five months	Patches on scalp, several along middle line, the rest unsymmetrical	Brown hair. Thin, healthy.
16	M.	boy	Two months	Three patches on scalp.	Brown hair. Healthy. Recovery after five months' treatment by Ac. Canth.
17	F.	8	Eight months	Patches on scalp	Also ordinary impetigo of face and scalp.
18	M.	26	Eight months	Scalp and moustache. Pubes, &c., unaffected	Strong healthy man, dark hair.
19	M.	9	Twelve months	Two patches on occiput.	
20	F.	5	Three months	Three coalesced patches.	
21	M.	21	Three months	Five patches on scalp.	
22	F.	22 ¹	—	Patches on scalp	Left arm and leg wasted from infantile palsy.
23	M.	12	Four years	Confluent patches over nearly the whole scalp. Eyebrows unaffected	Dark hair. It came on after chorea "from a fright." No rheumatism; no bruit.

¹ This young woman said that seven years ago she had the same bald patches

No.	Sex.	Age.	Duration of disease.	Present extent.	Remarks, &c.
24	M. ¹	11	A few months	Almost the whole of the scalp	A well-marked patch of tinea circinata on temple.
25	F. ²	11		Extensive baldness of scalp. Eyebrows and eyelashes also going	Healthy. Light hair.
26	M.	20		Patches on scalp	Cured with Ac. Canth.
27	M. ³	—		Patches on scalp (hair light)	Cured with Ung. Iodi.
28	M. ³	—		Patches on scalp (hair dark, almost black)	Cured with Ac. Canth. Hair first white.
29	F.	4	Several months	Extensive over scalp	Light brown hair.
30	M.	24	Five months	Numerous patches on scalp	
31	F.	21	Six months	Several patches on scalp	Fair.
32	M.	37	Second time	Three patches	Cured with Lin. Myrist.
33	F.	4	Five months	Three patches	Brown hair. Well nourished.
34	F.	10	Fourteen months	Two patches	Dark hair. Well nourished.
35	M.	5	Four months	Nearly the whole scalp	Fair hair. Healthy.
36	F.	10	Three months	Several large patches	
37	M.	18	Six weeks	One patch on occiput	Dark. Thin. Healthy.

on the head, and attended for two years at Guy's Hospital, at the end of which time she was cured.

¹ This boy's hair is said to have fallen off last summer and to have grown again.

² A sister is said to have lost her hair in the same way and to have recovered it.

³ These two men (Nos. 27 & 28) were between 20 and 30 years old.

ON THE FATAL TERMINATION OF DIABETES,

WITH

ESPECIAL REFERENCE TO THE DEATH BY COMA.

By FREDERICK TAYLOR, M.D.

WITHIN the last few years considerable interest has been excited in the fatal termination of diabetes by coma, and various attempts have been made to explain its occurrence by reference to alterations in the chemical or physical properties of the blood. I have had the opportunity of watching several such cases in the wards of Guy's Hospital, and I have collected the notes of them in the hope that they might form a contribution of more or less value to the subject, at least from a clinical and etiological point of view. I was also partly led to do this from the impression I formed that cases of death by coma were really very much more frequent than writers on the subject had formerly represented them to be; and I was further anxious to ascertain if the cases which did not die of such complications as phthisis, pneumonia, and gangrene and of intercurrent diseases, terminated in one and the same manner, or how often death occurred from simple exhaustion or any form of blood-poisoning apart from the death usually described as by coma.

During the last eight years, from 1873 to 1880 inclusive,

43 deaths have occurred at Guy's Hospital from diabetes out of a total number of 159 patients admitted. At the end of this paper I have appended the notes of these cases, either as complete histories of the illness, or as accounts of the fatal termination only. They have been taken from the inspection-books and the ward-books of the hospital. Of the 43 cases, a complete post-mortem examination was made in 37, in 1 the brain and spinal cord alone were examined, and in 1 the kidneys only. In four cases there was no autopsy.

TABLE I.—*Fatal Cases of Diabetes.*

Death by coma, no disease found post-mortem	.	.	14
„ no autopsy	.	.	2
Old phthisis, coma	.	.	3
Recent phthisis or pneumonia, coma	.	.	7
Ulceration of the bowel, coma	.	.	1
Pyelitis and suppurating kidneys, coma	.	.	2
Carbuncle, granular kidney, coma	.	.	1
Exhaustion (?), no autopsy	.	.	2
Phthisis	.	.	8
Pneumonia	.	.	2
Bright's disease and peritonitis	.	.	1
			—
			43

From this table it is seen that at least 30 cases, or two thirds of the total number of fatal cases died with comatose symptoms; that 10 died from complications, for the most part pulmonary, and without coma; while 3 cases must remain uncertain, chiefly from want of details in the reports sufficiently accurate to draw conclusions from.

The comatose cases require, however, further analysis, and divide themselves into several groups, according as the coma was the sole cause of death or was associated with one or other morbid condition of the viscera. There are, firstly, 14 cases in which post-mortem examination revealed no disease of any organ of sufficient gravity to cause death, or of a kind to cause coma; in many, indeed, nothing whatever. Congestion of the lungs is noted in some, in others coarseness or pallor of the kidneys, with excess of fat, and in 1 case a peculiar condition of the pancreas. These are all typical cases of death by nervous symptoms without phthisis or pneumonia, and they form one

third of the total number of deaths. Besides these there are 3, which should undoubtedly be classed with them, since the changes which were found post-mortem were inactive conditions of the lung of a phthisical nature, quite incapable in themselves of bringing about a fatal result in the way it actually happened. In one (Case 1) the right lung presented a caseous mass with pigmentation and calcareous matter; in another (Case 15) there was old phthisis with pleural thickening and pigmentation and caseous or calcareous nodules; and in the third (Case 16) a small patch of caseous induration undergoing calcification in each lung. The third division consists of 11 cases in which more or less active disease of different organs was found post-mortem; and amongst these are many in which it is not easy to determine whether the coma was dependent or not on the coexisting visceral lesion. Three had recent phthisis with cavities, 4 had pneumonia, 1 pneumonia with gangrene, 1 had ulceration of the small intestine resembling that of typhoid fever, and the remaining 3 had disease of the kidney, respectively, calculous pyelitis (Case 27), suppurative nephritis with dilated calices (Case 29), and granular degeneration (Case 29).

No doubt most of these conditions are sufficient causes of death, and would have been regarded as the actual causes if considered apart from the clinical history; but the question is whether their effects were not anticipated by the more rapid action of those changes which in the first group of cases were alone sufficient.

The case is analogous to that of poisoning by an overdose of morphia in the course of phthisis, typhoid fever, or other serious disease. The post-mortem examination considered alone would lead to the death being attributed to the fever or phthisis, and the share that the morphia had in it would only be recognised after inquiry into the clinical symptoms. The analogy is all the more close because the death by coma in diabetes resembles in many ways the effects of poisoning, and has been by many attributed to the presence in the blood of a special product of decomposition (aceton, acetonæmia).

Whether this be the case or not, the symptoms are to a certain extent characteristic, and their fatal effects are proved by such cases as those forming the first group. If, then, in

the course of diabetes they supervene upon some visceral lesion, such as phthisis, and lead as directly to a fatal termination, as they do when no phthisis or other lesion exists, then it is surely reasonable to regard them alone, and not the lesions found post-mortem, as the actual cause of death; and all the more if the visceral lesion in ordinary cases is not accompanied by any such clinical phenomena. Looked at from this point of view, there are some of these 11 cases which undoubtedly died of diabetic coma, though the autopsies alone might attribute the death to visceral complications. The most unequivocal is Case 24. A lad, *æt.* 14, had had diabetes for two years, and had been under treatment the greater part of that time. While still under the treatment and passing a large quantity of sugar, he was taken with abdominal pain and distension which were thought to be the result of indiscretions on Christmas day. There was no marked nor persistent elevation of temperature, no diarrhoea, and certainly nothing sufficiently characteristic to warrant a diagnosis of typhoid fever. In a few days he became drowsy and collapsed, with reduction of temperature to 96° , and presented the characteristic picture of diabetic coma or collapse. The ulceration of the ileum, whatever its origin, was clearly not a sufficient explanation of the mode of death. Cases 18, 19, and 22 had pretty extensive phthisis, but I have set them down as having died from coma, because they appear to have been seized in the course of the disease with a comparatively sudden development of coma, instead of continuing the steady down-hill course of phthisis in the ordinary way.

There remain the cases of pneumonia and the cases of renal disease, both difficult to estimate rightly, on account of the frequency with which they end in coma, independently of diabetes. Of the 4 cases in which pneumonia was found post-mortem, it appears to me that 3 may be regarded as belonging to the comatose cases. They are Nos. 20, 21, and 23. In the latter two, the development of the nervous symptoms was remarkably like what it is in typical cases, and the pneumonia was relatively slight in extent. In Case 20 the patient was admitted comatose, and no doubt of the connection with diabetes being entertained, the treatment by injection of a saline solution was attempted, though without success. It was only after death that the condition of the lung was ascer-

tained. In the fourth case of pneumonia terminating in coma, the physical signs of pulmonary disease, which proved to be caseous pneumonia with cavitation, had lasted more than two weeks, and coma supervened twenty-four hours before death. In the absence of any details as to the characters of the coma, I have preferred to consider this case doubtful, and do not include it among the cases of diabetic coma proper.

For similar reasons I have thought it best to place aside three cases with renal complications, though I think it is very probable that one of them (Case 29) did really die of diabetes independently of the chronic and apparently latent disease of his kidneys. Indeed, they are all recorded as "diabetic coma," which is at least an indication of the interpretation that was put on the symptoms by those who saw them at the time; but as the report of one case is unfinished and the histories of the other two are not very closely detailed, I cannot hope with the notes here appended to carry conviction to the minds of any one disposed to doubt their exclusively diabetic origin. As to the nature of the renal changes, there was in the first case reddening and ecchymosis of the pelvis of each kidney, with dilatation of the calices, suppuration of the parenchyma of one of them in lines along the pyramids, and depressed cicatrices on the surface of both. In the second the examination was incomplete, but as the patient had all the symptoms of renal calculus in addition to diabetes, and as Mr. Davies-Colley had actually extracted a calculus from the urethra, the kidneys were examined. Both organs were large, white, and flabby, and the right pelvis, in which was a calculus the size of a pea, was inflamed. In the third case, that of an old man who came into a surgical ward for a large carbuncle, the kidneys had a very granular surface, with wasting of the cortex and thickening of the arteries.

Out of the whole number of 43 cases, there are only 2 in which it appears that death took place from the simple exhaustion of diabetes, and without the occurrence of the condition known as diabetic coma. They are Cases 31 and 32. Unfortunately there was no post-mortem examination except of the nervous system in one of them, and this was carried out with reference to the question of microscopic alterations of the vessels and perivascular spaces in diabetes generally.

If my view of these cases has been correct, the following table shows the cases classified according to the actual or efficient cause of death.

Fatal Cases of Diabetes.

Coma	No visceral lesions	.	.	.	14
	Visceral lesions inactive	.	.	.	3
	Visceral lesions active	.	.	.	7
	No post-mortem	.	.	.	2
				—	26
	Coma (diabetic ?), renal disease	.	.	.	3
	Pneumonia, influence of coma doubtful	.	.	.	1
	Coma absent, no post-mortem	.	.	.	2
				—	3
	Phthisis, pneumonia, Bright's disease, peritonitis without coma	.	.	.	11
				—	43

Of course this list does not represent all the possible causes of death in diabetes. Diabetics are known to be susceptible to the influence of fever poisons, and one instance of this occurred some years ago in Guy's Hospital, when typhus was communicated by one patient to three others in the same ward; one of them had diabetes, and he died of the fever thus contracted. Gangrene of the extremities may also cause death, and moreover, diabetics may be attacked, like other people, by apoplexy, malignant disease, and the lesions prevalent with advancing age. This, at least, seems to me the only reasonable way to look at such cases as those mentioned by Lecorché,³ in which convulsions or paralysis occur in the course of diabetes,

¹ In the 'Guy's Hospital Reports' for 1875, vol. xx, Dr. Hilton Fagge made some remarks on the fatal termination of diabetes, and gave the statistics of the cases of which post-mortem examinations had been made in the hospital for the preceding twenty-one years, terminating with the year 1874. It will be seen that my analysis covers a portion of his. If I took only the six years, 1875 to 1880, in order to give results which might be taken with his, Cases 2, 5, 12, 23, and 28, amongst the comatose cases, and two others, would have to be omitted. The figures would then run as follows, and still present a large proportion of cases in which coma was the cause of death. Thus, coma with and without visceral lesions, 22; coma with renal disease, 2; doubtful cases, 3; phthisis, pneumonia, &c, without coma, 9; total, 36.

³ 'Traité du Diabète,' Paris, 1874.

in connection with cerebral softening and hæmorrhage. No doubt a larger number of cases would include some of these last instances; but the list may be accepted as showing that they are not very common, and that the chief causes of a fatal termination are phthisis, pneumonia, and the train of nervous symptoms usually known as coma. Pneumonia, apart from phthisis, occurred only in four cases, and three of these had also coma. These results are most strongly confirmatory of those given by Dr. Fagge in his article already alluded to, but I find that a much larger proportion of the deaths are due to coma, Dr. Fagge's figures being phthisis 21, pneumonia 11, coma 8, and sudden death, probably allied to the last, 2, out of a total of 45.

Hitherto I have spoken of "coma" and "nervous symptoms" without any qualification, but it will not be forgotten that in 1874 Kussmaul¹ described a "peculiar kind of death in diabetes" which he had seen in three cases, and which, though terminating in coma, was characterised by other symptoms as follows:—1. A dyspnœa of a peculiar kind, consisting in deep inspirations and expirations without cyanosis or œdema, the respirations being at the same time rapid and regular, but becoming slower in the stage of coma, and often accompanied during expiration by groaning. 2. Quickened action of the heart, with small, feeble, and regular pulse. 3. Great excitement, with groaning, restlessness, and violent pains. And 4. Coma. There can be no doubt that these observations of Kussmaul have directed a closer attention to the phenomena of death in diabetes, but, as might have been expected, it was soon pointed out² that the newly-described symptoms under the first three heads were more or less obviously present in many cases published prior to the date of his article, although those who reported them may have been content to emphasize only the coma, or the exhaustion, or the suddenness of a fatal termination. Indeed, I think it only wants a careful observation of a sufficient number of cases of diabètic coma to show that the kind of death described is by no means peculiar in the sense of being unusual, but is really only the most developed form of

¹ 'Deutsches Archiv,' 1874.

² Scheube, 'Archiv der Heilkunde,' 1877, p. 389.

the common mode of termination, when the patient does not succumb to visceral complications.

Of the cases here reported I find that more than twenty described as coma, or coma and collapse, clearly showed symptoms in addition which will allow them to be classed with the cases described by Kussmaul and by others after him. Such symptoms are the abdominal pains, the laboured sighing or heavy breathing, the restlessness with moaning, screaming, or groaning, semi-coma, from which the patient can be partially roused, collapse, pulselessness with violent action of the heart, dry skin and lividity from stagnating circulation. The end comes more or less rapidly in different cases, with more or less variation in the intensity of the different symptoms, with the predominance of coma in one group, of heart-failure in another, or of the respiratory peculiarities in a third. But from a consideration of the twenty-six cases I have grouped under the head of death by coma, I am inclined to think that no subdivisions can be made from a clinical point of view, but that all gradations exist between those cases which least resemble, and those which are the exact counterparts of, Kussmaul's typical instances.

Senator, in a recent article on diabetes,¹ seems to me to have adopted a similar view, including all such cases under the term "diabetic coma." He alludes to the early cases recorded by Prout, and mentions later authors, especially Kussmaul, as having given cases and descriptions. His own summary of the symptoms is exceedingly good, and is here transcribed for further comparison with my own cases. It is as follows :

"Sometimes suddenly without any premonition, sometimes after a first stage of agitation, with general uneasiness, oppression, anxiety, and pain in the region of the stomach, the patient becomes somnolent, moves about restlessly, generally groaning loudly. The pulse becomes frequent, the arterial tension is low, the breathing is hastened and deep, although there is no impediment in either the upper or lower portions of the respiratory apparatus. The extremities become cool, and even the general temperature of the body falls below the natural, and, finally, death ensues amid the deepest coma, sometimes after the supervention of twitchings."

¹ 'Ziemssen's Encyclop.,' Engl. ed., vol. xvi, p. 916.

These cases are also in accordance with the statement of Dr. W. Roberts, who says¹ that, "if diabetes does not terminate by one of its complications, the patient becomes gradually drowsy, and finally dies comatose."

I propose now to gather, from the cases at my disposal, such information as they afford with reference to the predisposing or exciting causes, and the chief clinical features of this mode of termination in diabetes.

Origin of the symptoms: pathogeny.—In considering what causes the outbreak of these symptoms many questions suggest themselves. Is it the final exhaustion of the nerve centres which have so long been failing in their functions? or is it an accidental occurrence, brought about by some external influence in a patient otherwise promising well, or at least stationary? Is it due to undue accumulation of a morbid product already in the blood? or to the new formation of some such body? or to the physical conditions of the circulating fluid?

Many views have been suggested. On the side of chemical changes, the view that the symptoms are due to excess of acetone in the blood (acetonæmia) has received support from many, while others consider them to be only a manifestation of anæmia. Among physical theories may be mentioned the suggestion, on which Dr. Hilton Fagge² founded an attempt to treat the patients by intravenous injection of saline fluid, that a thickened condition of the blood has been brought about by the drain of fluid through the kidneys. Another is the more recent view, put forward by Dr. Sanders and Dr. Hamilton,³ that the abundance of fat in the blood leads to fatty embolism of the capillaries of the lungs and other viscera.

To take first the aceton theory, the proof of its truth probably requires a good deal more than the clinical demonstration of the presence of aceton about the patient, since it has been shown that the mere presence of a small quantity of this compound in the blood is insufficient to cause symptoms of the kind under consideration. But the cases which have been observed with reference to this point at Guy's Hospital have

¹ 'Urinary and Renal Diseases,' 3rd edit., 1876, p. 231.

² 'Guy's Hospital Reports,' vol. xix, 1874, p. 173.

³ 'Edinburgh Medical Journal,' July, 1879.

given hitherto entirely negative evidence. It has been stated that such patients emit a strong fragrant smell, like acetic ether. I have never observed this myself, and in certainly two cases, Nos. 1 and 24, it was distinctly absent. In Case 13 a sweetish odour was noticed in the breath, and in Case 22 an odour like that of apples. Case 13 also had a sweetish odour like nitrous ether about him, but it is probable that in neither of these cases was it different from what occurs in the earlier stages of diabetes. Nor was there any odour of acetone about the blood or viscera in the two first-mentioned cases, although they were most typical instances of the form of death under consideration; but in Case 10 a sweetish odour was noticed when the body was opened, and in Case 7 a faint odour not unlike that of cats. In Case 13 the viscera were examined by Dr. Goodhart and Dr. Stevenson for the presence of acetone, but with a negative result.

The arguments in favour of an uræmic origin for the symptoms seem to have as little to support them. They are the resemblance of the symptoms in the two cases—the diminution of the quantity of the urine just before death, the occurrence of albuminuria, and the condition of the kidneys after death. There are no doubt many things in common in the two conditions of coma, and perhaps in neither case such complete constancy as would make discrimination always easy. But even supposing they are alike, that goes a very little way towards proving their origin from the same cause. The diminution of the urine has been frequently noticed, and I have little doubt that it is constant; albumen has also been present in at least seven cases. But I think it highly probable that one, if not both, of these symptoms, to which I shall again refer, are the result and not the cause of the nervous disturbance. As to the conditions of the kidneys, they were quite healthy in seventeen cases, and in the other seven, which make up the twenty-four, most typical and unequivocal cases, the changes were only slight, and the organs were described as "coarse kidneys," "large and coarse," "looking fatty," "pale and flabby, apparently fatty," &c. These are not conditions with which we commonly associate uræmic coma. As previously mentioned, I have grouped apart three cases in which there was pronounced

renal disease, but they form but a small proportion of the whole number of cases.

Coming now to the physical theories, the only evidence that these cases afford in reference to the theory of blood thickening is to be found in the results of the treatment attempted. Dr. Fagge's first case was partially successful; five hours after the injection of a saline solution into the blood, the patient, who had been comatose, with scarcely perceptible pulse, sat up in bed and answered questions. A case coming under my own care shortly afterwards was treated in the same way but without success. Five other cases have been similarly treated at Guy's Hospital, in only one of which was there the slightest improvement, and that for a few hours only.

Drs. Sanders and Hamilton, in support of their view that fatty embolism was the cause of the symptoms, point to the fatty state of the blood in diabetes, to the anatomical evidence of fat embola, and to the similarity of the symptoms with those of fat embolism after fracture. The fatty state of the blood has been noticed by other writers, and Dr. N. Moore showed a specimen of the kind at the Pathological Society in November, 1880, but it does not seem to be constant. In two of the present cases (Nos. 3 and 4) the blood is described as natural in appearance; in another (Case 7) it was fluid and appeared healthy under the microscope; and in another (Case 5) there was the usual decolorised clot in the right heart. In two cases a milky condition was present.

In Case 11 the left side of the heart contained ordinary pale fibrinous clot, but in the right side was "a large clot, the upper surface of which was milky white and of creamy consistence, utterly unlike ordinary fibrin clot. Its lower surface was soft and much resembled raspberry cream. It did not stain the endocardium; it seemed to cling to the fingers but did not stain them." It was also very unlike ordinary red clot. The upper part of this clot was found both by Dr. Goodhart and by Dr. Mahomed to contain a large quantity of free oil-globules.

In Case 6 the blood in all the veins, both of the root of the neck and in the inferior vena cava, was opaque as if mixed with pus and milk, and of a lilac or purple colour. When allowed to stand, a white opaque cream rose from it, and this was seen under the microscope to contain granular matter. The left

side of the heart contained natural-looking clot; the right side had a soft, milky-looking clot, and some liquid blood of the same peculiar appearance as that found in the veins.

It may be mentioned that the blood in this case had been treated during life by the injection of a saline solution, but it cannot be urged that the creamy appearance was due to this, since no mention is made of a similar condition in the three other cases so treated, and in a fourth (Case 7) it is expressly stated that the blood was natural in colour, and had no creamy layer like that present in the case of Ann E— (No. 6), which had occurred a short time before. This case occurred some years ago, and no search was made for fat embola.

In three cases the viscera were examined microscopically for fat embola with the aid of osmic acid staining, in two cases by myself, and in one by Mr. G. F. Crooke. In neither case was anything found.

The subject of diabetic coma is very fully discussed in a recent article by Ebstein,¹ who regards all these different theories as valid in different cases. He calls attention to the changes which he has found in the renal epithelium in cases of diabetes, consisting partly of a hyaline transformation, partly of a disappearance of the nuclei with breaking up of the protoplasm into lumps. He considers that this necrosis of the epithelium is due to the various conditions which have been recorded in published cases, viz. alterations in the amount of water in the blood and tissues, hyperglycæmia, acetonæmia, and the presence in the blood of acetic acid, alcohol, albuminates, oxalates, or of fat. Once produced, this necrosis acts prejudicially by preventing the proper elimination of the poisons which circulate in the blood-tissues as a result of the morbid metamorphosis, thus determining a condition analogous to, but not identical with, uræmia. He suggests, however, that a great accumulation of these poisonous products might exceed the powers of even healthy kidneys to excrete them.

The predisposing and exciting causes next claim our attention; they include the age and sex of the patient, the duration of the disease, and the influence of treatment, of local injury or lesions, and of fatigue.

¹ 'Deutsches Archiv,' Bd. 28, Heft 2 and 3, February, 1881.

Age of the patient.—It is well known that in patients who become diabetic at an early age, the prognosis is much more grave than in others; the patient is much less likely to be benefited by treatment, and the disease tends to run a rapid course. Of the fatal cases here recorded the greater proportion are under thirty years of age, and this result is largely due to comatose cases.

The following table will show the relations at once, the first half giving the total number of patients, the total number of fatal cases, and the twenty-six cases distributed amongst the several diseases from ten to seventy years of age; and the second half showing the percentages corresponding to the figures in the first. It will be seen that while of all cases those under thirty form 45 per cent., they form 53½ per cent. of the fatal cases, and 69 per cent of the comatose cases. The particular relation of youth to death by coma is best shown in the case of the first decade, where the difference in the percentage of the total cases and the total deaths is insignificant, but becomes at once considerable—an increase of 41 per cent.—on comparing the latter with those dying of coma.

Age.	Deaths by coma.	Total deaths.	Total cases.	Corresponding percentages.		
				Deaths by coma.	Total deaths.	Total cases.
From 10 to 20.....	5	6	24	19·2	14·	15·
20 to 30.....	13	17	48	50·	39·5	30·
30 to 40.....	5	10	35	19·3	23·2	22·
40 to 50.....	2	7	30	7·7	16·4	18·3
50 to 60.....	1	2	13	8·8	4·6	8·2
60 to 70.....	—	1	9	—	2·3	5·5
	26	43	159	100·	100·	100·

Sex.—The relations of sex to the fatal result in diabetes do not seem from these cases to call for any special remark. If it be attempted to compare the different sexes at different ages, the figures are so small that the results could not be depended on. It will be sufficient here to note that the 159 cases admitted comprised 114 males and 45 females, giving a percentage of 28 for the latter on the whole number; while the fatal cases numbered 32 males and 11 females, a percentage of

25.6 for the latter; and the 26 deaths by coma included 18 males and 8 females, a percentage of 30 for the females. The difference in these figures representing the percentages is so little that one may say they afford no real evidence that the female sex is more prone than the male sex to death from diabetes in general, or to the special mode of termination in coma. It must, of course, not be forgotten that the cases of patients who come to the hospital and go out partially relieved cannot be contrasted with fatal cases, as one could contrast cured and fatal cases of typhoid. That the cases die in the hospital must be regarded rather in the light of an accident than as a result of failure in the treatment, since those who go out probably die within a limited period either at home or in some other hospital, or in the workhouse infirmary. This rarity of cure in diabetes considerably lessens the value of any observations as to the preponderance of males or females among cases dying in hospital, so far as the disease itself is concerned; but a larger number of cases might give conclusive results as to the mode in which that death took place. These remarks apply also, though in a less degree, to the question of the patient's age.

Duration of the disease.—No period of the disease seems exempt from the liability to a rapid termination with nervous symptoms.

Some of the present cases ran rapidly to their end within a few weeks; in other cases months or years elapsed before they were carried off by coma. The analysis of the first twenty-six cases give the following results:

Duration of the disease.	No. of cases.
From 3 to 6 weeks . . .	4 (Nos. 4, 7, 9, 12).
„ 2 to 6 months . . .	6 (Nos. 2, 3, 8, 10, 13, 22).
„ 7 to 12 „ . . .	4 (Nos. 5, 17, 19, 25).
„ 1 to 2 years . . .	3 (Nos. 1, 16, 20).
„ 2 to 4 „ . . .	7 (Nos. 6, 14, 15, 18, 23, 24, 26).
Uncertain, of whom one perhaps only	
3 weeks . . .	2 (Nos. 11, 21).

It has been remarked that the mean age of these cases is represented by a very low figure, and it appears that some of the youngest cases have the shortest duration, the mean age of

the first group in the preceding table being only twenty-one years. In the succeeding groups the patients are progressively older and older, the mean age of the second group (death within six months) being twenty-five, and that of the third group twenty-eight. It is further remarkable that of 10 cases dying within six months, 9 belong to the group of cases in which no lesion was found post-mortem. To contrast with these, the cases which certainly did not die of coma may be taken, and of these, 2 died in from three to six months, 2 in from six to twelve months, 1 in from one to two years, and 5, or exactly one half, lasted more than two years. Any further analysis of this small list would be unprofitable. The conclusions seem to me that the majority of the rapid cases, or those terminating in less than twelve months, died of coma; that the majority of those dying of coma lasted twelve months or less; that the majority of those dying of chest disease or other complication alone lasted more than twelve months.

Influence of treatment.—As coma developed at almost all periods of the disease, so it took place under the most different conditions as to treatment. I should, of course, apply the word "treatment" here to nothing which did not include strict dieting. Of the 26 cases, 4 were never treated by diet at all; 8 had been treated with more or less success at some former period of the illness; and 5 were under treatment at the time. In the other 9 cases the information on this point is not sufficiently definite. I cannot make out positively that in any case the urine was entirely free from sugar when coma came on, though no doubt in some the disease had been more or less under control. For instance, in Case 1 sugar was present a few days before death, but the daily quantity of urine had for some time past been only two pints; in Case 24 also the quantity of urine was within the normal, though, it is true, there was abundance of sugar; and in Case 6 it was stated by the friends that the patient was able to do her work well, and was not troubled with polyuria, but the urine drawn from the bladder contained twelve grains of sugar to the ounce. The majority of those who had been under treatment formerly seem to have relapsed into their previous condition, and often to have relaxed in the strictness of their dieting. Case 14 was in the hospital five years before her fatal illness,

and left without any sugar in the urine; after three years she relaxed her diet, and was under a homœopath. Case 20 had left the hospital with the urine free from sugar; she kept to the restricted diet for some time, and then substituted German black bread for the gluten bread. There was a sudden aggravation of the diabetic symptoms with abundance of sugar in the urine. In one case the treatment was never successful in completely removing sugar from the urine, but the quantity had probably increased, as he had been attending regularly at another hospital for some time before his death.

Amongst the cases which died of chest disease alone, there are 6 in which some treatment had been attempted; 1 in which the history of treatment is doubtful; and another in which no mention is made of any treatment until admission, when the patient already had extensive phthisis. One case had the symptoms of phthisis for three months before those of diabetes.

From a careful review of all these cases, I should say that they afforded no evidence that either treatment or the absence of treatment determined death in one way rather than another.

Influence of local lesions.—I mean by this to inquire whether the coma is brought on by such a disturbance to the general health as would be caused by a slight blow or injury, by tonsillitis, abscess in the ear, or any of the minor ailments to which we are all more or less subject. Among the twenty-six cases I only find one in which such a connection should be traced; and here (Case 1) the patient suffered from headache for some days, and an abscess burst and discharged from the right ear the day before the serious symptoms occurred. Constipation was present in many cases, and might be regarded as having something to do with the onset. Of the severer internal lesions, such as were found in Cases 18 to 24, one would be the less inclined to regard them as determining the coma the longer their duration previous to it. Thus, whilst the pneumonia of Cases 20, 21, 23, may have had a share in its causation, this is much less likely in the case of the phthisis of Cases 18, 19, and 21, and the bowel ulceration of Case 24.

Influence of fatigue.—Several writers have noticed that the termination by coma or sudden collapse has been preceded by some unusual fatigue or exertion, and this connection was especially striking in the cases mentioned by Prout and by

Dr. B. Foster. The former¹ describes four cases of rapid death, and expressly states the individuals were in their ordinary state of health when they left home, and that their deaths could only be ascribed to the fatigue incidental to travelling. Dr. Foster's² cases are given in more detail. One was a man, aged thirty-seven, who was thought to be improving under treatment by lactic acid, and spent much of his time in the open air, "going daily in an omnibus to the outside of the town, where he took a short walk into the country, and then returned home by the same conveyance. On the day but one before he sent for me, he had walked farther than usual into the country, and had in consequence walked home a distance of some four miles. He arrived in a state of extreme exhaustion. The next day he felt extremely weak, and did not go out; he took some purgative medicine which did not act." Dr. Foster saw him the following day, when he presented the respiratory phenomena in a high degree; he became gradually drowsy and comatose, and died eighteen hours later, so that a period of nearly three days elapsed between the exertion and the fatal termination. In the second case a lady, aged twenty-nine, had suffered from diabetes about eighteen months. She had arranged to go a journey of about twenty miles by rail, and felt quite as well as usual on the morning of her journey, and quite equal, she thought, to the effort. She had some distance to drive to the railway station, the train was late, and she was fatigued by the extra time spent in reaching her friend's house. The following day, when Dr. Foster saw her, she had well-marked respiratory symptoms, and died twelve hours later. Dr. Fagge also³ points out that out of 8 cases that died of coma at Guy's Hospital in the years 1854 to 1874 inclusive, 5 died within five days of coming to the hospital, and he attributes the fatal termination to the fatigue and excitement which the patients underwent in connection with the journey. It is certainly quite remarkable what a large proportion of those who die comatose come to the hospital only a few days before their death. Of the 26 cases here analysed, only 4 had been in the hospital from one to three or four months, whereas 14, or more than half, died within a week, and

¹ 'On Stomach and Renal Diseases,' 1848, p. 83.

² 'British and Foreign Med.-Chir. Review,' Oct., 1872, p. 497.

³ 'Guy's Hospital Reports,' vol. xx, 1875, p. 180.

the remaining 4 in from eleven to sixteen days. But I shall shortly show that, though the actual coma may be of short duration, there are often other indications or premonitory symptoms of a crisis, which may be perceived a week or more before death; and I believe that in at least many of the cases that come in and die the journey does not stand to the death in the relation of cause to effect, but rather that the patients are prompted to seek admission by some sensation of illness and exhaustion beyond what we can perceive in them, or they themselves could describe.

In reference to the mere length of the journey, I find that out of the 14 cases who died within four days of admission, only 3 came from a greater distance than five or six miles. Two of these were already comatose and died on the day they were admitted, a result so rapid that it can scarcely be attributed to the journey, when the clinical history of other cases of diabetic coma is fairly considered. I am not, however, prepared to say that in these cases the patient may not have undergone some other fatiguing exertion. More than half of the 14 patients came from distances of three miles or less.

Onset of the symptoms.—I have already said that this is by no means always sudden, or even rapid. The semi-comatose or fully comatose condition is often preceded for some days by an unusual condition of ill-health; and the patient may present in this early stage, which begins from six to nine days before death, one or more of the following disturbances:—Unusual weakness or exhaustion, loss of appetite, unusual constipation, slight drowsiness, breathlessness, headache, sleeplessness, pain in the abdomen, epigastrium, or loins. There is, of course, little that is characteristic in these symptoms, except in such as are really the beginning of the developed condition; still the occurrence of many of them together should excite apprehension. On looking carefully over the cases, I think it can be said that the fatal termination was foreshadowed by indications of this kind, commencing as early as four days before death in 3 cases, five days in 3, six days in 2, seven days in 2, and eight days in 1.

The final stage of which the picture has been already given in Kussmaul's and Senator's descriptions, may develop very rapidly out of these preliminary symptoms. Patients who

have only been observed to be more than usually weak and exhausted, or have failed in their appetite, are suddenly seized with laboured breathing or drowsiness, and sink in a few hours. There is, however, no one of these cases in which the coma has come on as absolutely sudden unconsciousness, nor has there in any case been a fit; and more commonly the symptoms may be said to have begun from one to four days before actually terminating in death. As an instance we may take Case 9, a boy aged fifteen, who had only had symptoms of diabetes a few weeks. When admitted on the 18th of the month he was greatly exhausted, had deep and sighing respiration, and complained of sinking at the pit of the stomach. On the morning of the 21st he was much collapsed; later in the day he was drowsy, but could be roused, and he died, in spite of a saline injection into the blood, on the morning of the 22nd. Case 2 was drowsy and listless, with loss of appetite on the 4th of the month, less drowsy the following day, walked up the ward complaining of pain on the morning of the 6th, but in two or three hours more was lying in bed, not easily aroused to answer questions, breathing heavily with loud moaning expirations, and died in the course of the afternoon. Case 1, after some days of headache and constipation, during which an abscess discharged from the ear, was taken with sudden abdominal pain on the 13th of the month, which continued on the 14th, with sighing breathing, quick feeble pulse, depression of temperature, the patient still conscious when aroused, but becoming less and less observant of things going on around her. She died in the course of the following day.

In Case 15 the patient had griping pains in the abdomen with vomiting and retching on the 2nd of the month; then a restless night, getting out of bed frequently; on the 3rd he became semi-comatose, with deep breathing, weak pulse, cold extremities; on the 4th he tried to pass water and failed, at midnight was insensible, but restless, and died on the 5th in the early morning. A more rapid course of the comatose symptoms occurred in Case 17, where a patient a few days after admission had facial neuralgia, accompanied by pains down the back and unusual weakness, with failure of the appetite. At 7 p.m. the severe pains in the back and epigastrium necessitated the application of poultices, and an hour later he was

found suddenly gasping for breath, like an asthmatic, with cold extremities, pinched face, small pulse, nausea, and still acute pain at the epigastrium. From this time he gradually became drowsy and comatose, and died at four in the afternoon the next day.

Pain.—A point of considerable interest is the occurrence of more or less violent pain in a great number of cases. Kussmaul called attention to this. Of his cases one had "violent pain in the hypogastrium," and another had pain "arising in his inflamed stomach," as well as "pains in the hips and hypogastrium." A large number of patients in my list have suffered from this symptom, and so severe was it in some cases, and so suddenly developed, that it was, in combination with the collapse occurring at the same time, taken as evidence of a probable gastric or intestinal perforation. Case 1 was of this kind, and the pain was situated about the umbilicus and round to the back. In Case 2 there was great pain all over, and hot poultices were applied to the abdomen; and in Case 8 there was great pain in the abdomen, and "it was thought she must have peritonitis." Seven others had pain described as abdominal or "pain in the bowels," but of these, one is the patient that had ulceration of the ileum, and his abdomen was at the same time somewhat distended and tympanitic. Four had pains in the stomach or epigastrium, but as one is described as griping pain, it was probably not confined to the epigastric region. The others had respectively "sinking at the pit of the stomach with constriction across the chest" and "pain on pressure over the liver and below the ribs." Altogether sixteen had this symptom of pain, which was sometimes accompanied by retching or actual vomiting. None are recorded to have had pains in the hips. In the majority nothing was found after death which would explain it; the abdominal viscera were normal. But in a few there were changes which might be regarded as having some connection with the pain, though perhaps not as the causative conditions. It is, for instance, curious that in the two cases in which the resemblance of the symptoms to those of perforation was most marked, there should have been found recent intussusceptions of the small intestines. I have never seen any statement as to the frequency with which these are seen in post-mortem examinations, but Dr. Goodhart tells me that they are by no means common; and the question

naturally arises whether the pain has been the result of an excessive peristaltic action of the small intestines, or whether the pain has originated in some considerable irritation of the sympathetic nervous system, of which irritation the intussusceptions are further evidence.

In two other cases it is noted that there were scybala in the rectum, and it may be observed that constipation is often amongst the early phenomena of this mode of death. In one of these the large intestine was much distended, the transverse colon was arched downwards, so that the whole abdomen was filled by it and the cæcum and sigmoid flexure. In the rectum were hard scybala, pushing out the perineum. In two other cases, the condition of the peritoneum suggested early inflammation, in Case 23 were some marks of recent peritonitis, and in Case 21 the small intestines looked injected and vascular, and a little viscid fluid could be obtained on scraping the surface. The intestines here also were loaded with scybala. This closely resembles a case recorded by Dr. B. Foster.¹ "The peritoneum covering the intestine and forming the omentum was seen to be minutely injected, the small capillaries being filled with red blood. There were no adhesions connected with the peritoneum, and no fluid in the cavity." This case, however, as well as these two of mine in which this injection was observed, had inflammation in the chest, either pneumonia or pleurisy. In Case 24 one could not dissociate the abdominal pains from the ulcerative lesions in the small intestines. Within the last few weeks, and since I began the analysis of these cases, there occurred a typical case of diabetic coma under the care of Dr. Moxon, in which during the stage of semi-coma there was considerable tenderness of the abdomen. If the surface was at all pressed upon she moaned, and put up her hands to prevent it. The abdominal aorta was here pulsating most violently and out of all proportion to the apparent impulse of the heart; the abdomen was not distended, but rather empty, and the sensation of tenderness seemed to be connected with the condition of the aorta. The same pulsation was present in the femorals and radials, and a sphygmographic tracing showed the almost monocrotous type of the pulse of hæmorrhage or unfilled vascular system.

¹ Loc. cit., p. 492.

I have said this occurrence of pain is of interest, but it is also of considerable importance to know the connection of these pains with the coma or collapse of diabetes, and for this reason: that if an opiate is given to subdue the pain, the comatose condition which supervenes may be wrongly attributed to the drug, either by the physician or by the patient's friends. I remember this question, whether the drowsiness was natural or due to opium recently administered, to have been raised in two cases not included in the present list. One, a boy aged nineteen, who came to the hospital some years ago, had some opium given him just before undertaking the journey to the London, was already drowsy when he arrived, and died in the course of twenty-four hours. The other, a gentleman aged thirty-five, who had been under my treatment some months, was seized one night with colicky pains, which became so severe in the morning that he sent for the nearest medical man, and was treated with opium and an enema. The bowels were opened, but he subsequently became unconscious, and died during the following day. He had the deep sighing breathing, restlessness, and groaning, seen in diabetic coma, and though, as in the other case, the suspicion was entertained for the moment that the opium treatment might have been at fault, it was exonerated after a full consideration of all the circumstances.

A few points of interest remain. The urine is reported in some cases to have diminished in quantity with the onset of the severe symptoms. In cases such as Nos. 2 and 3 where death took place seven or eight days after admission, it is difficult to say how much of this was due to the treatment instituted and how much to the commencing failure of the functions of the body. But in Case 13 the diminution of the urine was noticed early before admission, and such a diminution is at once explained by the relation of the urinary flow to the force of the circulation. One of the characteristic features of diabetic coma is the condition of collapse, with extreme feebleness of the circulation, and the reduction of pressure in the kidneys results in a smaller quantity of urine, which may be at first mistaken for actual improvement. At the same time the quantity of sugar is less.

The urine contained albumen in seven cases; "some," "a

little," "slightly albuminous," in three cases; a trace in two cases; one-fifteenth in another; and in the last as much as two thirds the quantity of urine. In one only of these cases was the albuminuria noticed before the comatose symptoms came on, and in one it was the result of examination of the urine taken from the bladder after death. In three of these cases the kidneys were healthy, in two fatty, in one large and coarse, and in the last the calices were somewhat dilated, but without evidence of inflammation of the organs. This was the case in which the albumen was most abundant. Albuminuria is of course not rarely a complication in diabetes; but it seems not impossible that the passage of albumen might be a direct result of the coma and collapse, especially as in one of these cases the duration of the disease was only a few weeks, and in three others was four months or less; but nearly all of the cases were admitted with the coma present or immediately threatening, and no opportunity was afforded for ascertaining the condition of the urine just previous to the severe symptoms.

The condition of the pupils does not appear to be either constant or in any way remarkable. In four cases they were observed to be contracted, in three dilated, and in two of medium size.

Treatment.—The more certain we become of the frequency of coma as a termination of diabetes the more hopeless does its treatment become, and certainly the cases that have occurred at Guy's Hospital give but little encouragement to the physician who is called upon to deal with such a case. Dr. Gamgee, however, alludes to a case in which the symptoms were recovered from, though they returned again some time after. The only treatment besides that of stimulants (administered either internally, or as brandy by subcutaneous injection), that has been at all freely tried at Guy's Hospital, has been the intravenous injection of water or saline fluids above alluded to (p. 141); the slight amount of success obtained in two cases has been there described. The fluids used were, in one case, water alone; in three cases a mixture of sodium chloride and sodium phosphate; and in two cases the solution used by Dr. Barnes for injection in puerperal cases, consisting of sodium chloride two drachms, sodium phosphate six grains, sodium carbonate fifteen grains, and potassium chloride twelve

grains, in two pints of water. The quantities injected ranged from twenty-six to forty-seven ounces.

The cases which follow are arranged according to the classification adopted in the table on page 152. Eleven of the cases have appeared in previous volumes of these 'Reports,' and the shortest possible abstract is given of them, with a reference to the paper in which they were first recorded.

FATAL CASES OF DIABETES.

Death by coma, without visceral lesions.

CASE 1.—Hannah E—, æt. 28, was admitted, under Dr Pavy's care, November 4th, 1879. Her parents are alive and well. Two brothers have died of phthisis, and two sisters, aged 14 and 34, of diabetes. Two other sisters are alive and well. When nine or ten years of age she had scarlatina, followed by acute desquamative nephritis and dropsy, the latter continuing more or less up to the present time. She has had no other illness. A little more than twelve months ago she first noticed herself becoming very thirsty. She went on for five months without saying anything about it, and then, consulting a medical man, was ordered pills and a special diet. She has continued this to the present date, with the exception of substituting toasted brown bread for the gluten variety. She got much worse from the commencement until March last, but then improved, and has gained flesh again up to the present time.

She has a flushed, somewhat anxious face, light hair, grey eyes, waxen and moist skin. She passes urine frequently to the extent of ten or twelve pints in the day; it has a specific gravity of 1040, contains much sugar, and no albumen. The tongue is a dark colour, bright at tip and edge, papillary plainly marked. Teeth bad. Appetite good. Much thirst. Bowels always confined. The abdomen is tense, apparently swollen, but is not œdematous, and containing no fluid. The liver hard, rather enlarged. Spleen normal. Lungs normal. Heart normal. Pulse strong, regular, quick, incompressible, and rather persistent. Temp. 99°; pulse 96; resp. 19.

On November 5th the usual diet was ordered, with gluten, bran, or almond biscuits, and the following pill was given three times a day.

R Codeia, gr. $\frac{1}{4}$.
 Ext. Nux. Vom., gr. $\frac{1}{4}$.
 Ext. Aloes, gr. j.
 Ext. Lactucæ, gr. ij. Fiat pilula.

10th.—Doing well; codeia increased to one third of a grain.

17th.—Codeia increased to half a grain.

24th.—Complains of indigestion and flatulency, with pains across the chest and swelling of the abdomen.

During the month of December the urine measured from two to three pints, and contained from 2000 to 2700 grains of sugar in the twenty-four hours. The bowels were often constipated. In the beginning of January, 1880, the bowels were much confined, and she had headache. The constipation was met by the use of enema saponis, and by increasing the aloes and decreasing the codeia in the pill, but the headache was worse until an abscess developed in the right ear, and discharged on the 13th of the month.

During the night of the 13th she complained of severe pain in the abdomen, and was utterly collapsed, so that brandy had to be administered. On recovering she still complained of the pain, which was diffused, extending round to the back, and worst just below the umbilicus.

On the 14th, at 10 a.m., the face was flushed, the pulse could not be counted at the wrist, and was only occasionally felt. At 12 noon it was small, thready, about 96 in the minute; the temperature was 98·2°; resp. 26. She was lying on her back with the legs drawn up, frequently groaning, occasionally rolling over on to the side; countenance anxious; breathing difficult.

I was now in charge of Dr. Pavy's wards, and I saw her myself at 2 p.m.; she was then complaining of pain in the upper part of the abdomen, between the umbilicus and the sternum, and pointed to the sacrum as the seat of the pain in the back. She was conscious and rational, and partly turned over to show me where the pain was. The abdomen was tense, but not distended, generally resonant. The breathing was deep, scarcely sighing, but as if forced. The pulse

was small, thready. Pupils rather contracted. I ordered her beef tea, brandy and egg, by enema; and only a little ice by the mouth. She had been thought by the house-physician to have acute peritonitis from perforation, but I felt confident myself that this was only the termination of diabetes.

At 9.30 p.m., I saw her again; she was lying on her back, the head and chest slightly raised. Temp. 97° ; pulse 141; resp. 30. The breathing was now very much deeper, and much more closely resembled that of diabetic collapse—a deep inspiration, forced expiration, and then a short pause. The pulse was perhaps slightly fuller than at 2 p.m.; the hands and fingers cold and slightly livid; the tongue brown and dry; pupils contracted. She was still conscious and answered questions, but for the most part was unobservant of what was going on around her. The face was somewhat dusky, thinner than it was a few days previously, when she considered herself well. Ordered,

R Sp. Æth. Sulph., ʒss.

Ammon. Carb., gr. vij. Aq. ʒj, 4tis horis.

The bowels had been open twice on the 13th and once this morning; the last motion was soft and brown in colour.

On the evening of the 13th she vomited and again at midday of the 14th, bringing up thirty to forty ounces of mixed fluid and solid of a brown colour.

In the course of the following day she died without any essential alteration in the symptoms.

Post-mortem.—Lungs rather congested; no signs of commencing phthisis or other lung trouble. In the small intestine were two small intussusceptions, evidently quite recent, if not post mortem; when pulled out it could not be seen where they had been. The kidneys weighed 9 ounces, and looked healthy; there was no shrinking of the cortex; the stellate veins on the surface were plainly marked. Liver to all appearance healthy. Brain, heart, stomach, and spleen healthy.

CASE 2.—Sarah B—, æt. 22, admitted February, 27th, 1874. Duration of symptoms four months, more marked the last two months. No history of treatment. Dieted on admission. Very weak and exhausted on March 2nd. Bowels constipated.

Drowsy, and listless on March 4th. Severe abdominal and general pains on the 6th, coma with heavy breathing and moaning expirations; death in the evening. Treatment by saline injection into the veins. Post-mortem: organs generally healthy, kidneys fatty, granules in renal epithelium, stroma healthy. (Reported in 'Guy's Hospital Reports,' vol. xix, 1874, p. 521.)

CASE 3.—William C—, æt. 25, was admitted under Dr. Wilks in January, 1880. He is a clerk, and has had no illness before. The symptoms of diabetes commenced three months ago, and on admission he passed nine pints of urine daily. He was treated by dieting, codeia, and mineral acids. The daily quantity of urine was seven or eight pints in March and April, six or seven pints in the early part of May.

On May 20th "Patient is much more drowsy than he was, and rather dispirited." May 23rd: has lost about four pounds in last two weeks; no change in the lungs. May 26th: no lung mischief, no dropsy, can see perfectly well. May 27th: ordered to bed, being ill and feverish. May 28th: became very much worse towards the evening and fell into a comatose condition at nine o'clock; extremities cold; breathing rapid; patient was lying with his mouth partly open, eyes half closed, pupils dilated and equal. May 28th: is in the same condition, cannot be roused; breathing heavily, 30 a minute. Water was injected into the veins; it roused him somewhat, and he recognised his father about fifteen minutes after the injection. The temperature at 3 p.m. was $100\cdot6^{\circ}$, at 6 p.m. $103\cdot2^{\circ}$. He had slight rigors after the injection, about two o'clock. He died at nine p.m. Post-mortem: the organs were all healthy and the blood had its natural appearance.

CASE 4.—Charles C—, æt. 27, admitted May 15th, 1876. Symptoms of diabetes observed six weeks ago; appears not to have been treated; appetite failed two or three weeks ago, and bowels much confined lately. He is now emaciated, and already semi-comatose; restless, often dozing off, but can be roused to answer questions; violent action of heart; feeble, rapid, small pulse; deep sighing respiration; cold surface, with stagnating circulation. Continued restless during the night;

died the following morning. Post-mortem: organs generally healthy. The cortical part of the kidneys yellow and fatty. (Reported in 'Guy's Hospital Reports,' vol. xxii, 1877, "On the Nervous System in Diabetes," by Drs. F. Taylor and Jas. F. Goodhart.)

CASE 5.—James M—, æt. 38, admitted under Dr. Fagge, February 14th, 1873. Duration of symptoms nine months; had never been treated. He gave up work on February 8th; on the 12th his appetite failed; on the 13th he was delirious. Comatose on admission, with scarcely perceptible pulse, and slow laboured breathing. Saline injection into veins, after which the pulse was stronger, and he sat up to answer questions, and took his medicine. Continued much the same during the 15th, but in the evening relapsed, and died about 3.30 a.m. on February 16th. Post-mortem: organs healthy; kidneys rather large and coarse. (See "A Case of Diabetic Coma" by Dr. Hilton Fagge in 'Guy's Hospital Reports' vol. xix, p. 173.)

CASE 6.—Annie E—, æt. 35, was first admitted into Guy's Hospital in February, 1875, and then gave a history of two year's thirst and free urination; later on she had excess of appetite. She was two months in the hospital improving under diet and treatment. She went out and was able to do her work well, and was not troubled by polyuria.

On Sunday, May 16th, her friend left her well, and had a telegram at three o'clock on Tuesday, the 18th, to say she was ill. He found her at 4 p.m. insensible; after much brandy she recognised him; she last spoke on Wednesday, the 19th, at 2 a.m. and was admitted into Guy's at midnight. She is well nourished and "does not look diabetic." The face is flushed, the skin warm. She is in deep coma; the pupils equal, contracted, perfectly insensible to light. Breathing a little stertorous, respirations deep and regular, and the nostrils dilated. Temp. 97.5°; pulse 118; resp. 25; pulse small and feeble. Urine drawn off, sp. gr. 1015, acid, clear, pale, sweet, contains albumen two thirds, and of sugar twelve grains to the ounce. Brandy roused her a little; she made a few reflex movements with her mouth. At 4 p.m. a pint of Barnes' saline solution was injected, and at 8 p.m. a second pint. An enema of beef tea,

egg, and brandy was also administered, but she died at 9.45 p.m. without recovering consciousness.

Post-mortem, by Dr. Fagge.—Fairly nourished; a little œdema of the ankles. Head not examined. Lungs healthy, except a little œdema. The posterior part of the right lung, at its base, was marbled of a greenish-brown colour, and at first I thought it was gangrenous; but it subsequently appeared clear that the colour was due to the entrance of gastric contents after death. Larynx: discoloured with gastric contents (there had been no vomiting during life). Heart, left side: natural-looking clot. Right side: soft milky-looking clot and some liquid blood, having the peculiar purple milky appearance of the blood in the venous system generally.

Veins: in all the veins, both of the root of the neck, and also of the inferior vena cava, the blood had a most remarkable appearance. It was opaque, as if mixed with pus and milk, of a lilac or purple colour; and looked exactly like the pulpy liquid which sometimes oozes from a soft spleen. When allowed to stand a white opaque cream rose from it. This under the microscope showed granular matter. The blood-corpuscles were even (not crenated); it was thought they looked enlarged, and I certainly could not make out that they were smaller than the leucocytes, which were visible in the field of the microscope; but the magnifying power was insufficient to enable one to speak positively.

Ileum: solitary glands rather distinct; no tubercles. Liver healthy, 66 oz. Spleen healthy, 6 oz. Kidneys 12½ oz. appeared rather coarse, otherwise healthy; some of the calices distinctly dilated and the pyramids flattened. Bladder markedly hypertrophied; the bands on its inner surface much more visible than usual, and its muscular wall decidedly thickened. It was estimated as one sixth of an inch thick, the bladder being by no means closely contracted.

CASE 7.—George K—, æt. 18, was admitted under my care on May 26th, 1875. He was well until three weeks ago, when he had frontal headache, and soon afterwards noticed the usual symptoms of thirst, polyuria, and emaciation. On admission he is much wasted and exceedingly weak; weight 6 stone 8 pounds; skin dry and harsh, with mottlings of greyish-brown

colour covering the forearms, thighs, and legs, and the chest and upper arms to a less extent. Tongue dry and red, with yellow fur; pulse feeble, 86; urine of specific gravity 1032, containing thirty grains of sugar to the ounce. Chest: left side slightly less resonant; hollow expiratory sound above the left clavicle; jerky inspiration below. Heart and liver normal. Abdominal aorta pulsating strongly. Bowels regular, but the motions have been dry. The appetite, at first voracious, has been failing lately. On May 28th he was ordered the special diet, and codeia. The following day he was tending to become comatose; he could hardly talk or sit up in bed; there was pain on pressure in the right hypochondrium, just below the ribs. Pulse 120; temp. 97.4° ; resp. 16, sighing. Tongue dry; thirst and hunger abated; pupils contracted. A soap enema brought away some lumpy feces, and he vomited twice.

On May 30th, continued stupor, with the conjunctivæ still sensitive, deep breathing, and feeble pulse. Temp. 94.8° ; pulse 124; resp. 29.

A saline solution was now injected into a vein as follows:—Ten ounces at 10 a.m., seventeen ounces at 10.50 a.m., and twenty ounces at 6.50 p.m. No good result followed, and he died at 10.30 the same night. The post-mortem examination was made by Dr. Goodhart, and is recorded as follows:

“Coarse features and coarse brown hair; fair muscular development; no fat; no dropsy; no spots or scars. During life some brownish spots had been noticed, but the only evidence of any skin eruption now was the livid mottling from local stasis in the capillaries. Cranial bones normal; dura mater and sinuses, &c., the same. Brain, 51 oz., firm, rather full of blood, and of dark colour; convolutions healthy; pons and medulla healthy. Pleuræ healthy. Lungs congested; blood in them rather treacly, no phthisis. Heart weighed $7\frac{1}{2}$ oz., presented the spiral form, muscular fibre good. Both sides a small amount of clot of ordinary character, i.e. black in the dependent parts and fibrinous at the upper. The right heart on being opened *in situ* allowed some thin serous fluid to run away, but there was apparently no more than the serum from which the blood had settled. The blood was generally fluid in all the veins of the neck, &c., and in the viscera.

“I examined the blood microscopically, and it looked in all

respects healthy. It had none of that swollen glassy appearance of the corpuscles seen in a former case of diabetes treated by injection of a saline fluid. Vessels good; both the veins into which the fluid had been injected looked red and ecchymosed in their lining membrane above the puncture, and that on the right side had a small clot in it of normal appearance.

"When the body was opened it was noticed that it gave off a peculiar odour, not unlike that emanating from cats, in a faint degree.

"Liver 49 oz., blood fluid. Gall-bladder contained good bile; section very homogeneous, and wanting in lobulation; dark coloured, like a child's liver. Pancreas healthy; spleen healthy, 4 oz.; kidneys healthy, $7\frac{1}{2}$ oz."

CASE 8.—Mary L—, æt. 30, was admitted under Dr. Moxon on April 17th, 1875. The symptoms began about Christmas time, with excessive thirst and hunger, cramps in the legs, and wasting. She gave up work a fortnight ago. On admission she was passing ten pints of urine; the heart and lungs were normal. On April 20th she was ordered half a grain of codeia three times a day. During the next few days the urine diminished in quantity, but she afterwards became comatose, and died in about three days.

At the commencement of the severe symptoms (on April 28th) she had pain in the abdomen, and it was thought she must have peritonitis. Her hands were not cold, and her pulse, though feeble, could be felt.

Post mortem.—Brain healthy, firm; pons and medulla appeared to the naked eye quite healthy. Lungs healthy; they emitted a fair quantity of blood on section, and one of them some frothy fluid; no tubercle. Liver, 51 oz., appeared healthy, it had not the diabetic odour. Stomach and intestines healthy, but there was an intussusception of the jejunum. Bladder not hypertrophied; it contained a little creamy fluid exactly like pus, but this was proved microscopically to be bladder epithelium detached. Fallopian tubes adherent to the ovaries; the left one closed and distended into a cyst. Kidneys rather congested, 10 oz.; no appearance of being fatty.

CASE 9.—Frederick P—, æt. 15, admitted under my care

September 18th, 1876. Symptoms began only five weeks ago; he has lost flesh and strength rapidly, and the bowels have been very confined. Complaints of sinking at the pit of the stomach; feeble pulse; deep, sighing respirations. Viscera apparently normal. His collapsed condition increased, and on the 21st he became drowsy, with livid surface and cold extremities. Saline injection into the veins; death on the 22nd at 9 a.m. Post mortem: organs healthy, the kidneys containing an excess of fat. (Reported in 'Guy's Hospital Reports' vol. xxii, 1877, loc. cit.)

CASE 10.—Sarah R—, æt. 20, was admitted under the care of Dr. Pye-Smith, August, 25th, 1879. She had not been perfectly well since Christmas, and about two months ago there was an alteration in her manner and temper, she became silent and sullen, and her appetite became ravenous. Three weeks ago she had jactitation of the right arm and leg, which was not constant, and was never observed during sleep. Hunger and thirst increased and she lost flesh. On admission, there was a sweetish odour of the breath; the urine was abundant, of sp. gr. 1045, pale and saccharine. During the next few days the quantity of urine fell from five pints to three. She complained of severe cramping pain in the abdomen and epigastrium about September 3rd, and this was relieved by an enema. On the evening of the 4th the pain returned with increased intensity, and she rapidly became comatose, with slow, sighing respiration, small feeble pulse, and cold extremities. She died about 11 a.m. on the 5th. The post mortem was made by Mr. G. F. Crooke.

Reddish hair, freckled face; anæmia; emaciation; deficient development of mammæ and pubes. Brain healthy; the cord showed nothing abnormal to the naked eye. Lungs and heart small, weighing together $23\frac{1}{2}$ oz., healthy. Nothing abnormal to note about the abdominal viscera, except that all evolved a more or less sweetish odour, being quite warm when removed. Liver, rather pale, yellowish and mottled in places, otherwise healthy. The lungs, liver, and kidneys were afterwards examined microscopically for fatty embolism, but with a negative result.

There were embola in all the pulmonary arteries composed

of red and white blood cells; no fatty particles. The epithelium of the kidney tubules, especially of the tubuli recti, had undergone well-marked fatty change; no fatty globules in the blood-vessels. The liver looked fairly healthy as regards cell structure; the capillaries here and there between the cellular network seemed abnormally widened and in many cases filled with blood cells and leucocytes.

CASE 11.—Alfred S—, æt. 40, was admitted in October 1880. He had come from the country to consult Dr. Pavy for diabetes. He fell suddenly ill, and was brought to the hospital with the commencement of coma, in which he died. When seen by the house physician, Mr. Howell, at nine in the evening he was just sensible and could answer questions; the pulse was about 160, the breathing was quick; later on it became deeper. He was quite comatose at 1 a.m. and died at five. The post mortem was made by Dr. Mahomed.

Well formed, plentiful rather grey hair; thick beard; well nourished. Head and spine not examined.

Thyroid rather large, structure appeared normal.

Lower lobes of both lungs very congested and greatly softened. They broke down very readily, and had a strongly-marked raspberry cream appearance.

In the middle mediastinum on left side, between the pericardium and pleura, and encroaching on the left lung, was a nodule the size of a walnut; on section this proved to be an encapsuled cheesy and creamy mass of pus, probably the remains of a suppurating gland.

Bronchi slightly congested; the small ones contained a good deal of frothy serum.

Heart: muscular fibre good. Left side contained ordinary pale, fibrinous clot. In right side, a large clot (? two or three ounces); its upper surface was milky white, and of creamy consistence, utterly unlike ordinary fibrin clot; its lower surface was soft and much resembled raspberry cream. It did not stain the endocardium; it seemed to cling to the fingers but did not stain them. It was unlike ordinary red clot and much less like fibrinous clot.

Abdomen generally normal. Liver, average size. Its lobules were well marked out, but there did not appear any

increase of fibrous tissue. Gall-bladder normal, with healthy bile.

The pancreas presented a peculiar appearance; it was rather larger than usual; on its surface, and also scattered about the gland between the lobules, was a milky-white, fatty-looking material, as it were smeared over it; this appearance extended into all the adjacent fat, notably into the transverse fissure of the liver, and into the upper part of the mesentery; it presented the appearance of the fatty matter seen in atheroma. The lymphatic glands in the neighbourhood of the pancreas and in the transverse fissure were rather enlarged, and on section appeared of a pale yellowish colour, somewhat cheesy looking, but perfectly homogeneous.

Spleen soft, with much of the raspberry-cream appearance.

Kidneys of fair size, not much enlarged considering the size of the man. Structure seemed normal, perhaps somewhat infiltrated by fat.

Bladder normal; not perceptibly thickened; rugæ below mucous membrane not particularly well marked, though just visible.

The upper part of the blood-clot was found, both by Dr. Mahomed and Dr. Goodhart, to contain "a large quantity of free oil globules."

CASE 12.—George E. S—, aged 25, was admitted on March 24th, 1874. He is a letter carrier and shoemaker, and has been married six weeks. He was always temperate and never had venereal disease. On March 8th, he felt ill, was thought to be suffering from a cold, and complained of thirst. Soon were observed polyuria, debility, and wasting.

On March 21st, the urine was found to be saccharine. On this day his appetite failed and he got rapidly worse. Micturition continued frequent. Diet was not restricted. The bowels acted regularly.

25th.—Present condition: height 5 ft. to 5 ft. 1 in., weight 7 stone; he used to weigh 10 stone. Face pinched and hollow, extremities cold, muscles flabby. He complains of aching pains all over him and breathlessness on the least exertion, and these symptoms have made their appearance since admission.

The chest is pigeon-breasted. The breathing is rather

laboured, but there is nothing abnormal in the respiratory murmur in front or behind. Expansion and resonance are good.

There is a mitral systolic murmur audible behind and in the axilla, but only indistinctly in front as a roughness of the first sound.

Abdomen : no pain nor tenderness ; liver and spleen normal. He is intensely thirsty, mouth dry, tongue thickly furred and yellowish. Urine 13 pints 14 ounces in twenty-four hours, slightly acid, sp, gr. 1018 ; sugar 32 grains to the ounce.

Head : no pain nor delirium ; sight clear ; optic discs healthy. There is a slightly sweetish odour about him like nitrous ether. Last night temp. 97.9° ; pulse 108 ; resp. 22. Getting drowsy this afternoon ; a blister was applied to the back of the neck.

26th.—Restless and wandering during the night. He was sick, and was noticed to have some difficulty in swallowing. Now he is collapsed, with cold surface, especially at the extremities ; ears blue. He lies semi-comatose, with eyes closed, can be easily roused, and appears to understand what is said to him, though he cannot answer intelligibly ; probably this and the difficult deglutition are due to the parched condition of the mouth. He is still restless, but makes no complaint. He has passed 1 pint 9 ounces of urine since yesterday afternoon. Much trouble to get him to take any fluid nourishment. Breathing laboured, but air enters freely all parts of the lung, and there are no abnormal sounds in any part. The resonance is good. Temperature low, resp. 24 ; pulse 65 ; very feeble. Fluid ran from his mouth, and brandy and hot drinks introduced by the stomach-pump failed to rally him. The restlessness increased, he passed his urine in bed, and at 6.10 p.m. turned on his bed, vomited, gave two or three convulsive gasps, and died. The post mortem was made by Dr. Goodhart.

Features sharp ; brown hair ; small make ; muscular. Brain, 51 oz., rather firm, but in all respects healthy. Cord examined carefully throughout ; at one or two places the vessels or spaces appeared large, but these were mostly in the grey commissure or the region of the central canal, so that it was doubtful if they were really abnormal. The substance of the cord seemed quite healthy.

Both lungs somewhat silky and failing to collapse; lung tissue healthy; rather tough at the apices; and all one lung œdematous. Bronchial tubes congested; blood not thick.

Heart, 12 oz., contracted muscle firm. Coronary arteries good. Uvula œdematous, no other evidence of inflammation about the throat.

Liver, 53 oz., with a peculiar smell, which could hardly be described as sweet, about its section; capsule thin; organ of a dirty red colour; section very homogeneous, very tough, and of an olive-brown colour. The blood expressed from the hepatic vein gave no reaction when tested for sugar.

Gall-bladder nearly empty; perhaps 1 drachm of bile. Spleen $8\frac{1}{2}$ ounces. Cervical ganglia looked healthy.

Kidneys, 11 oz.; capsule adherent but not thick; very firm on section. Section showed thick cortex, in which the striæ were very well marked, but in which a closely-set arrangement of minute dots was to be seen, such as is to be noted in an acute disorder of the kidney. What was, however, especially noticeable was that apart from this state it differed in the great facility with which the two parts could be separated from each other by the eye, and some of that blurred state existed, evidencing disease of stroma as well as of tubes microscopically; fat in the epithelial tube cells. The stroma very little fatty, and practically healthy. A mere trace of albumen in the urine from the bladder.

CASE 13.—John S—, æt. 35, was admitted March 19th, 1879, with diabetes of two months' duration. He was emaciated, the cheeks pale, without flush. Chest fairly well formed, mobility equal on the two sides; respiration abdominal and thoracic; resonance good in front and behind; breathing almost bronchial at right apex behind; sweetish odour of breath. The urine, which was seven or eight pints a month ago, is now between four or five pints, of sp. gr. 1037, containing much sugar, but no albumen, blood, indican, nor peptone.

March 22nd at 3 p.m.—Feels sick this afternoon since dinner; bowels not opened since 14th; soap enema opened bowels at 8 p.m. producing loose and thin lumpy stools. 10 p.m. pulse 112, very feeble, and collapsing. Temp. 97.5° in axilla, resp. 40 and laboured. Great weakness and tendency

to sleep. Face much drawn, pale and cadaverous. Complains much of feeling cold, and slight pain, but no tenderness in the epigastrium; constant desire to go to stool. Gradually sank and died at 11 p.m. The post mortem was made by Dr. Goodhart.

"The body when opened exhaled a little of a peculiar mousy odour that I have observed not infrequently in diabetes, but there was no smell of acetone about it.

"The brain weighed 46 oz.; it was firm and I examined it and the cord in all directions, and could find nothing whatever abnormal. The other viscera were quite healthy.

"The heart weighed only 8 ounces, the kidneys 11 ounces. The bladder was not hypertrophied. The liver was small, and homogeneous on section."

Some of the viscera were handed to Dr. Stevenson for the detection of acetone. Dr. Goodhart distilled some, but failed to recognise acetone in the distillate.

CASE 14.—Alice H—, æt. 18, was admitted January 7th, 1879. The symptoms of diabetes were first noticed in 1874, and she had been dieted with much relief both at home and in this hospital. Any relaxation of the diet aggravated the symptoms, and they had been increasing considerably prior to admission.

On admission she was much wasted, with enormous appetite and great thirst; the bowels regular; chest normal; heart's impulse in fourth space, first sound feeble; urine abundant, sp. gr. 1028, containing no albumen but twenty grains of sugar to the ounce. Mental faculties clear; pain in the lumbar region and between the shoulders, which she had had for three weeks.

On January 11th she complained greatly of pain in the abdomen, sides, and back.

13th.—The pains were still severe. The patient was very weak and could not walk without assistance.

14th.—At 2 p.m. she became delirious, and gradually sank into a semi-comatose condition. The pulse was imperceptible in the radials, it was beating 50 per minute in the carotids; the skin was cold. She died at 1 p.m.

Post mortem.—Lungs a little œdematous, otherwise healthy. Heart healthy; blood in the heart and vessels had the usual appearance; it was not lilac coloured as described in aceto-

næmia. Liver dark coloured, otherwise healthy. Kidneys pale and flabby, apparently fatty. Bladder markedly hypertrophied.

Diabetes ; old phthisis ; death by coma.

CASE 15.—Daniel D—, æt. 25, was admitted on August 2nd, 1876. The first symptoms appeared four years ago, and he has attended for treatment both at Guy's and St. Bartholomew's Hospitals. Since July 28th he has passed scarcely any water, and has had headache and giddiness, and has felt very weak. He is anæmic, with light brown hair, light eyes, cold and dry skin. He is constantly retching, and complains of griping pains in the epigastrium, but he has not been sick. He has headache, and feels very giddy. Tongue dry and brown ; brown sordes on lips and teeth ; no blue line. Extensors of hand not weakened ; sensation in upper limbs not diminished. Liver not observed to be enlarged. Spleen not felt. The chest appeared normal, with the exception of some prolonged expiration. Heart-sounds feeble.

August 3rd.—Yesterday complained of severe griping pains in the stomach and vomited. During the night has not slept at all, and has been retching, but has not vomited. Gets out of bed frequently. Has passed about two ounces of water, which is pale yellowish green, of sp. gr. 1028 ; it gives a slight opalescence on the addition of nitric acid, and contains twenty-one grains of sugar to the ounce.

4th.—Has passed a more quiet night, being semi-comatose, with deep breathing ; the eyes are turned upwards ; the face, yesterday pale, is now flushed ; weak, fluttering pulse ; extremities cold. He appears moribund. Warm fomentations have been applied to the abdomen ; he tried to pass water and failed. Bowels not open.

5th (12.15 a.m.).—Pulse 114, resp. 28. Insensible but restless ; moves the arms about ; conjunctivæ not quite insensible, pupils of moderate size, equal, hardly act to light. Transfusion of thirty-two ounces of a saline mixture ; then the pulse became 120 and fuller, but he died at 4.30 a.m.

Post mortem.—Brain appeared healthy. Lungs contained an excess of fluid. At right apex some old phthisis, with

pleural thickening and pigmentation; some caseous and calcareous nodules; no tubercles. Liver rather hard. Kidneys large and coarse, weighing 18 or 19 ounces. Bladder showed distinct hypertrophy of its muscular coat.

CASE 16.—John W—, æt. 29, was admitted on July 19th, 1876. Symptoms of diabetes had existed sixteen months. He was very much wasted, and on July 28th was very weak.

31st.—Chest resonant; heart sounds normal but feeble. His speech this day was slow and hesitating, and he appeared to be drowsy, lying down on his bed but not actually sleeping. The following day he could hardly get up stairs after being down in the grounds. In the evening he complained of pain in the stomach, became comatose at 8 p.m., and died at 7 the next morning.

Post mortem.—At the posterior part of the right upper lobe was a patch of induration, which was caseous, and in some parts undergoing calcification; the patch was not larger than a hazel nut. At the extreme apex of the left lung was a similar lump, the size of a walnut; it was caseous in some parts, and in others more or less calcified. All the other viscera were healthy.

CASE 17.—John W. B—, commercial traveller, unmarried, æt. 25, was admitted under Dr. Moxon on September 24th, 1880. He had scarlet fever ten years ago. Suddenly, in January last, he observed that he was getting thinner; noticed in March, that he had hunger and could not hold his water all night. Extreme thirst in May; he was then dieted. He has had no cough and no hæmoptysis. The viscera appeared quite healthy. Six weeks ago was passing eight or nine pints. The only pain of which he has complained has been down the front of the chest, and he has not got that now.

September 27th.—Was ordered almond bread, meat, milk, green food.

30th.—Some neuralgia to-day over right face, pains down the back, and feels great weakness. He is therefore lying on his bed instead of getting about the ward as usual. In the morning he was suffering from toothache, pain in epigastrium, sickness; and he had not had his bowels open for three days. After colocynth and mercury he had a slight solid motion, but

no relief from the other symptoms. Appetite is very bad to day; he ate a very small chop for dinner, however. Face somewhat relieved by painting with tincture of aconite. Dr. Moxon ordered tincture of gelseminum internally. At 5 o'clock he ate his tea and was about the ward. When the clinical clerk saw him at 6 p.m., he said he thought he should get some sleep, having had none the previous night. At 7 p.m. he was found complaining much of pain in the back and epigastrium, and hot fomentations were applied, by which he expressed himself as much relieved. Then at 8 p.m. he was found suddenly gasping for breath, and the house physician was sent for. He found the patient sitting on the side of the bed battling for breath like an asthmatic, with cold extremities, pinched face, small pulse, complaining of most acute pain at the epigastrium and of nausea. Air was found entering both lungs freely; the heart's action fairly quiet, pulse 120, resp. 28. No tenderness in the abdomen; fair pressure relieved the pain, which was all referred to the epigastrium. An injection of Morph. Acet. gr. $\frac{1}{3}$, and Atropin $\frac{1}{150}$ was given. Fifteen minutes later he was well enough to lie back in bed, having previously sat up to get breath. At 10 p.m. he was sleeping, and could be easily roused; pulse 120 smaller, heart's action much more violent. The patient, however, said he was much easier; but his breathing had the same characters. He was evidently sinking into a semi-comatose condition.

October 1st.—At 1 o'clock he was more comatose; at 5 o'clock a bad attack of breathing came on. At 2.50 p.m. the breathing was shallower and he was pulseless. At 4 o'clock he died.

Post mortem.—In the anterior part of the right upper lobe, just below the apex, was a caseous mass, the size of a marble, with pigmentation around, and with a little calcareous mass in its centre. No definite tubercles existed, but the affection was not altogether old, for a little opaque, puriform matter, welled up, perhaps out of bronchial tubes, from the margin of the mass at one spot. Larynx healthy. Heart: right side, much fibrin and blood of normal colour. Intestines healthy. Rectum contained much scybalous faecal matter. It was distended rather than contracted. Liver healthy. Kidneys coarse and fatty looking. Bladder reticulated by hypertrophy of its muscular coat.

Diabetes ; recent phthisis or pneumonia ; coma.

CASE 18.¹—Ebenezer F—, æt. 81, was under Dr. Fagge on August 2nd, 1876. He had had symptoms of diabetes two years, has recently suffered from diarrhoea, and three weeks ago noticed cough, with night sweats, and increasing emaciation. He has never been treated. On admission there were physical signs of phthisis at both apices, most extensive on the right side. The urine contained thirty-six grains of sugar to the ounce. Temp. 101·4°, pulse 120, resp. 32. He was ordered cod-liver oil, cinchonine, and the restricted diet.

On the 14th he was drowsy, and continued to be so until the 18th, when whilst sitting in a chair he appeared to have been taken with serious symptoms ; he was moved into bed and died in a few minutes.

Post mortem.—The lungs at the upper part were both extensively diseased, but it was at once evident that the disease was different from the ordinary pneumonic phthisis of diabetes. There was great increase of fibrous tissue bands intersecting the lungs, and there were numerous scattered cavities and clusters of yellow tubercles in all parts of the lung. Many of the vomices had marked caseous walls. Dr. Fagge was unable to identify any grey tubercle. Bronchial glands swollen ; one or two of them contained distinct small caseous masses. Larynx healthy. Intestines generally healthy, but in the upper part of the jejunum a single transverse ulcer with indurated edge, and a little subserous tubercle. In the ileum one or two caseating solitary follicles.

CASE 19.—Lucy F—, æt. 28, was admitted October 2nd, 1878, in a semi-comatose condition, with rapid, sighing breathing, and evidence of pneumonia. Symptoms of diabetes commenced in February ; in March she was treated in Guy's Hospital, and went out relieved in July. No history can be obtained of the beginning of her present symptoms. She died the day after admission.

Brain, 45 oz., looked perfectly healthy, as well as the spinal

¹ 'Guy's Hospital Reports,' 1877, loc. cit.

cord and medulla, and the vessels compared favorably with those of a non-diabetic nervous system.

Recent pleurisy in lung of right side. Left upper lobe was in a state of entire disorganisation, large ragged cavities filled with red grumous but inoffensive fluid. In the parts where the disease had not broken down it was a grey hepatisation in colour, but wanting in the solid feeling that is usual in an ordinary pneumonia. It was more like what might be supposed to be the condition in a rapidly disintegrating cedema. The patches were, however, well circumscribed. Dr. Goodhart remarked that it would be incorrect to call this pneumonia, of which there was very little. The tissue perished without the formation of inflammatory products.

The right lung had patches of broncho-pneumonia like the left. Larynx healthy. Heart, 6 oz., healthy. Liver, 69 oz., soft not abnormal; no unusual smell about these or any other parts. Kidneys, 14 oz., open textured, but not unhealthy. Bladder, no marked hypertrophy.

CASE 20.—Harriet H—, æt. 48, admitted December 16th, 1875. Symptoms had existed fifteen months and had been much relieved by treatment twelve months ago. Cough and emaciation the last six weeks. On admission, drowsiness, and complaint of pain in the left chest. Became comatose; a saline fluid was injected, but she soon died.

Post mortem.—Pneumonic patches at both bases, the left base partly gangrenous. Kidneys yellowish, and renal epithelium fatty; other organs healthy. (Reported in 'Guy's Hosp. Rep.,' vol. xxii., 1877, loc. cit.)

CASE 21.—John M.—æt. 24, was admitted under Dr. Pavy's care January 11th, 1876. The history was given by a friend, who says he was well till three weeks ago. He then caught a violent cold, and had a great deal of coughing; he never spat any blood. He has lost much flesh during this time. On January 6th he complained of severe pain in his bowels, which has continued ever since, becoming worse. His bowels have not been opened since that day, but were regular till then.

He is undersized, much wasted, looks very ill, and complains

of much pain, which he refers to his stomach, but which, he says, he sometimes feels in his bowels and sometimes in his chest. Heart normal. Lungs: at bases there is some dulness, with mucous râles occasionally. Liver and spleen normal. Fulness on the left side of the abdomen, probably fecal. Tongue dry and brownish red.

The patient is very restless, and does not seem more than semi-conscious. He requires a good deal of rousing to make him attend to any questions, and to them generally he utters incoherent replies. It is impossible to get any definite information as to his habits from him. There is no history of syphilis. The pain he has had since the 6th, has been, he says, aggravated by castor oil.

9.30 p.m.—He was very restless during the evening, but is quieter now. He seems quite unconscious. He has not passed any water since admission. The bladder is distended up to the umbilicus. Between three and four pints of urine drawn off.

January 12th, 10 a.m.—Almost pulseless, unconscious, moribund. Pupils widely dilated. Urine sp. gr. 1030. By boiling and adding nitric acid, it shows about $\frac{1}{15}$ th albumen, and there are about 19 grains of sugar to the ounce. Death took place shortly after noon.

The post mortem was made by Dr. Fagge.

Nourishment not evidently below the usual range.

Brain, 52, healthy, so far as could be discovered. No cavities visible in the pons or medulla. Pleuræ: some adhesions at the base over the mucous cyst, to be described presently, and the corresponding part of the inner part of the left lung was consolidated, and of a creamy-yellow, granular, dry appearance. It was tolerably firm on pressure, and very decidedly limited by the lobular markings. It was exactly like what the apex of the lung is often seen in diabetes—the “pneumonic phthisis” of that disease. Under the microscope the pneumonia proved to have the characters of croupous pneumonia, the alveoli stuffed with inflammatory products, some fibrillated lymph and granular matter, and a large number of leucocytes.

Peritoneum. Dr. Fagge was unable to satisfy himself whether peritonitis was commencing or not. The small intestines, though not distended, yet looked injected and vascular, and there were some doubtful suction-lines. Scraping the parietal

peritoneum with a knife, one got off a little viscid, glutinous fluid, not evidently opaque. Just above the diaphragm, pressing on the œsophagus and against the lung, was a cyst, the size of a pigeon's egg, containing an opalescent, jelly-like substance. Intestines healthy, no tubercle. Cæcum much dilated and distended with gas. The rest of the large intestines loaded in many places with scybala. Liver and spleen healthy. Kidneys, 14 oz., decidedly fatty. All the tubes under the microscope found to be black with minute fatty granules. The cells cohered well together in long cylinders; many of them were scattered loose about the field. No decided evidence of catarrhal swelling or multiplication of the epithelial cells could be found. The bladder was dilated, not hypertrophied.

CASE 22.—John W—, æt. 21, was admitted July 5th, 1880. Had good health till four months ago, when he was invalided from the army for diabetes. All the symptoms of diabetes were noticed by his friends. Five days before admission he was taken suddenly worse; had to take to his bed. There has been noticed a slight cough of late, never any spitting of blood. Yesterday he was wandering in his mind; to-day he is admitted unconscious.

July 6th.—Quite comatose, incapable of being roused; milk put into his mouth is barely swallowed; he appears to be moribund. He breathes regularly and deeply. Both apices resonant; expiratory sound is audible at both apices. Heart sounds faint. Pulse 100, full, regular. Temp. 99°. Pupils contracted. Breath sickly, and like the aroma of apples; this odour is perceptible as far off as the next bed. No urine has been passed since admission (12 hours). Bladder distended. Urine 1021, acid, slightly saccharine, slightly albuminous. He died on July 6th. The post mortem was made by the house physician, Dr. W. H. White.

Lungs: some phthisis at both apices, with small cavities; no tubercles to be seen; in various parts of the lung a few hard patches of a yellow colour, the largest about half an inch in diameter, probably inflammatory, but possibly infarcts; some pleuritic adhesions. All the other organs are healthy to the naked eye and not much congested.

CASE 23.—James H. B—, æt. 50, was admitted under Dr. Pavy, November 11th, 1874. He had had symptoms of diabetes three years, and appears to have had no treatment except the advice from a medical man that he should live well. He has been alternately better and worse. He now passes six or seven pints of urine daily, with more than 2000 grains of sugar. He was treated with the special diet. There was some cough, and moist sounds were heard over the front of the left lung.

December 3rd.—There is still a troublesome cough with a great deal of phlegm. Complains of pain in his abdomen.

4th.—The pain in the abdomen is gone, and the cough is better, though it troubles him at night. He has no appetite, refuses his meals, and takes only liquids. Pil. Doveri, gr. v, t. d. s. Effervescing saline mixture.

6th.—Suddenly worse; sleeping and lying in a drowsy condition.

7th.—Patient lies in a comatose state; will speak if roused sufficiently; says he feels in a state of stupefaction. Pulse very feeble; breathes heavily. His bowels have not been open since the 2nd.

8th.—Patient died quietly at 5.45. He was sick slightly before death, bringing up some dark-coloured fluid.

Post mortem.—Emaciation. No dropsy. Head not examined. Right pleura, general adhesions; at lower part of posterior surface a hard patch, with a collection of partly cheesy, partly calcareous matter between the layers of the pleura (evidently a dried up empyema). Lungs: all the posterior part of the left lower lobe in a state of grey hepatisation; the upper lobe quite free; a little recent pleurisy of the surface of the affected part of the lung. No phthisis in either lung. Heart, 12 ounces, healthy. Some atheroma of the mitral valve. Many adhesions between the liver and the stomach, and also between the spleen and the diaphragm. Liver, 56 ounces, appeared quite natural. Spleen soft, 9 ounces. Kidneys, 12 ounces, rather rough on the surface; a cyst, the size of a nut, containing a cheesy material, in the cortex. Bladder much hypertrophied; its muscular fibres forming raised bands interlacing in all directions, the mucous membrane protruding in numerous sacculi between these bands of muscular fibres.

Diabetes ; coma ; ulceration of ileum (typhoid ?).

CASE 24.—Ernest B—, æt. 14, was admitted October 21st, 1878. Occupation, newspaper boy.

Family history.—Both parents alive and healthy. Brothers and sisters healthy.

Has had measles, but in all other respects has been healthy since his birth.

Present attack.—Patient fell into the river two years ago and received a severe fright. He went to school for a fortnight after this, and then became seriously ill and took to his bed. He complained of severe thirst, passed large quantities of water, and had pains in his head and legs. He was treated by a medical man, who said he had diabetes. In about six months he became convalescent. Since that time he has had three relapses, and each time the symptoms have been more severe. A month ago he again became ill, suffering from thirst and passing large quantities of urine.

On admission he has a hot, dry skin, and flushed face. He cannot read with comfort, and the left pupil is contracted. The tongue is dry and slightly furred, the bowels regular, but appetite bad. He vomits occasionally, and has intense thirst and violent pain in the abdomen, which keeps him awake at night. The urine measures ten pints in twenty-four hours, is pale, of sp. gr. 1041, and contains 39·99 grains of sugar to the fluid ounce. Resp. 22, pulse 108.

23rd.—Urine 132 ounces, sp. gr. 1035, sugar 34·26 grains to the fluid ounce ; 4522 grains in the day.

24th.—Urine 210 ounces, sp. gr. 1037, sugar 7194 grains.

28th.—He began diet yesterday ; has been very sick this morning ; violent pain in head and abdomen. Ordered opium gr. $\frac{1}{2}$ and Ext. Nucis Vomice gr. $\frac{1}{2}$, t. d. s.

November 1st.—Complains of great pain in the head, but he is up and able to walk about the ward.

15th.—He is stronger, has gained in weight, and can go up and down stairs without difficulty.

December 4th.—Is daily about the wards, has a good appetite, and generally a feeling of thirst. The urine has a light

yellow colour, sp. gr. 1033, no albumen, abundance of sugar. The quantity of urine, which was ten pints and six pints on admission, fell rapidly to the normal limits; and from October 28th to December 12th, the average of the observations was two pints six ounces, the minimum being one pint two ounces, the maximum two pints eighteen ounces. During the same time the sp. gr. ranged from 1027 to 1038, and the daily quantity of sugar from 500 to 1700 grains, only on one occasion exceeding 2000, and once 1900 grains.

27th.—Complains of slight headache; he has not slept well the last two nights. There is a faint sickly odour about his breath.

29th.—He is in bed this morning; his head is bad; he has no appetite. The breath has a sickly sweetish smell. He explains his symptoms by saying that he had some plum pudding and other luxuries on Christmas-day.

31st.—He is no better; the tongue is slightly furred; the appetite bad; bowels open. Temp. 99.9° ; pulse 84; resp. 26.

January 2nd, 1879.—Is only passing about a pint of urine in the twenty-four hours. Complains of great pain in the head and abdomen. The abdomen is distended and tympanitic. Temp. 100.9° ; pulse 104; resp. 30.

3rd.—He was suddenly taken ill this morning between 12 and 1 a.m. His breathing is heavy and laboured; he is propped up with pillows, his arms extended over his head, the eyes prominent and staring. He complains of pain about the region of the heart; he answers questions readily. At 9 a.m. his temperature was 96.8° ; at 10 a.m. 96° ; at 10.30 a.m. 95.8° ; pulse 140; resp. 32. A castor-oil enema was given, and brought away a few small lumps of fæces; this was followed by some castor oil internally.

4th.—He has passed a very bad night, with laboured breathing. This morning he is not conscious; lies in a semi-comatose state, with eyelids half closed and eyes fixed. Temp. 98.8° ; pulse 146; resp. 28. At 1 p.m. temp. 99.6° ; the pulse cannot be felt; resp. 34. At 6 p.m. temp. 100.3° ; resp. 30. At 11 this morning ten minims of brandy was injected subcutaneously, and again at 12, 3, and 5, with marked improvement in the condition of the pulse; a few teaspoonfuls of milk and brandy have been taken.

5th.—Much weaker this morning. Lips pale and covered with dry scales. Radial pulse imperceptible. Nothing abnormal in the chest. Good pulsation in the carotids. Temp. 97.7°; resp. 34. At 12.15 noon, ten minims of brandy were injected subcutaneously, and fifteen minims at 1, 2, and 4 o'clock. He gradually sank and died at 9.10 p.m.

The following is Dr. Goodhart's account of the post mortem:

"Brain: membranes all normal except that they were excessively vascular. The pia mater everywhere minutely injected. I do not attribute any significance to this, as the boy died in a comatose state. The brain was firm but in all respects quite healthy to the naked eye. I searched very carefully through by slicing, but could find nothing in the way of gaps or pigmentary deposits visible to the naked eye either in the cortex, the cerebral ganglia, the pons, medulla, or corpora dentata cerebelli. The spinal cord was injected on its surface like the brain, but its substance was everywhere healthy.

"Lungs and heart healthy. Peritoneum healthy.

"The lower half of the small intestine contained numerous ulcers; they were mostly circular, as large as a threepenny piece, some larger, some smaller, and much more numerous near the ileo-cæcal valve than higher up. I did not at first take them to be typhoid ulcers, because they were one and all so flat, hardly raised at all above the mucous surface, and unaccompanied by any widening of their edges. They were all black in colour, and all occupied the whole or part of a Peyer's patch. At the lower part no healthy, or even unulcerated, patch remained, but higher up some of the patches were uniformly swollen by a thin layer of whitish or yellow deposit, and the intermediate plaques were in a state of partial ulceration, some of the deposit being still unsoftened; the rest of the mucous surface was injected, but otherwise healthy. But in the duodenum the mucous surface was studded over with irregular ecchymosed and very superficial ulcers, to which the term "hæmorrhagic erosions" would well apply. The solitary glands low down appear to have escaped, and there was no disease whatever in the colon. The mesenteric glands were much swollen, very soft, ecchymosed, and juicy. The pancreas and supra-renals looked quite healthy. Liver large, but looked healthy.

"Spleen large and brick-red in colour. The Malpighian corpuscles swollen and pulpy; all the tissues rather soft. Sympathetic nerves all looked quite healthy. Kidneys rather large, but quite healthy. Bladder closely contracted, and I think the muscle was thicker than normal for a boy of his age.

"Some adductor muscles were examined microscopically for Zenker's change, but none was found."

Diabetes; coma; no post-mortem examination.

CASE 25.—George G—, æt. 19, was admitted under Dr. Habershon's care, 31st October, 1876. Clerk. No intemperance; no venereal disease; no recollection of any injury to head or spine. Diabetes commenced last November or December, and in December Dr. Pavy restricted his diet and ordered codeia; he improved, and thought himself cured in April, 1876, but again got unwell a month ago. He lost flesh, had polyuria, thirst, ravenous appetite, and thinks his memory became weaker.

On admission he is tall, thin, fair, intelligent; height 5 ft. 9 in., weight 8 st. 6 lb. Skin dry, and inclined to peel; face florid now and habitually. Tongue dry, red, clean. Heart and lungs normal.

Abdomen distended and tense, $32\frac{1}{2}$ inches round the umbilicus. Some tenderness over the stomach and at other parts of the abdomen. Bowels inclined to be confined. Liver normal. Enlarged gland in the left groin. Urine, sp. gr. 1035; sugar 32 grains to the ounce. Ordered codeia and nux vomica.

November 1st.—Is very comfortable.

2nd.—Gets up in the evening. Bowels opened by castor oil. Passed nine pints of urine.

3rd.—Passed a restless night; and in the morning was noticed by the nurse to be very heavy, and scarcely intelligible in what he said. On replacing him in bed, he fell back heavily as though unable to support himself, and his breathing became laboured and the extremities cold.

9.30 a.m.—Patient on his back, breathing in a sighing manner though quietly; nothing like dyspnoea; is quite unconscious of what may be passing around; though he can be roused at times

and then appears to understand what is said to him; and can be made to drink. Extremities cold. Temp. 94.4°, pulse 74, fairly good. Pupils natural. One minim of croton oil was given him this morning, but it has had no effect. One pint of urine has been drawn off; it is of sp. gr. 1020, and contains 20 grains of sugar to the ounce. The quantity since 10 a.m. yesterday is 2 pints 9 ounces. Sinapisms to calves of legs; hot water to the feet; spirits of nitrous ether internally.

3 p.m.—Patient much the same; a turpentine enema has been given and has returned. Ordered—

℞ Ammon. Carb. gr. x.

Ex. aq. ℥ij. 4tis horis.

℞ Ol. Crotonis, ℥j. Statim.

He remained much the same till the morning of the following day, when the coma deepened and he died at 10 a.m. The purgatives never acted. There was no post-mortem examination.

CASE 26.—George S—, æt. 38, a bricklayer, was admitted October 3rd, 1877. Symptoms of diabetes had existed two years. He had had a chancre ten years previously, and had drunk freely of beer all his life.

On admission he had a feeling of weakness and dull aching pain in the lumbar region, and occasionally severe shooting pains in the head. The complexion was florid, and the face constantly flushed. He was alternately drowsy and restless. The breathing was not obstructed, but he took very deep inspirations, and the movements were mostly thoracic. The lungs appeared to be healthy. Heart normal. Pulse 80, weak but regular; temperature normal. Bowels now confined, though generally regular. Urine highly saccharine.

Was ordered restricted diet. The quantities of urine passed on the 4th, 5th, and 6th October, were respectively five, eight, and five pints. On October 8th he was very drowsy, and complained of pain in the abdomen and diarrhœa. Urine passed in the preceding twenty-four hours measured four and a half pints. On the following day he was very drowsy, and complained much of the pain; it was very difficult to get correct answers to questions put to him. The diarrhœa was still severe; the urine four pints. Temperature 98°. On October 10th he

was much worse, semi-comatose, and died at 8 o'clock in the afternoon. There was no post-mortem examination.

Diabetes ; coma ; renal disease.

CASE 27.—Charles P—, æt. 25, was always well until he had gonorrhœa six months ago. Came as an out-patient here six weeks ago, and was treated for gonorrhœa and orchitis; as he got well he had the first symptoms of diabetes, with polydipsia and polyuria. On January 2nd was passing five or six quarts, with much sugar, daily, and had then one grain of codeia, three times a-day. On January 9th the codeia was increased to two grains, and he appears to have improved.

On admission, January 11th, he looked fairly healthy. No excessive hunger. Urine sp. gr. 1035, containing sugar, but no albumen, though sometimes viscid mucus is passed with it. He was treated with opium, carbonate of ammonia, and dieting, and passed during the next three weeks five or six pints of urine of sp. gr. 1025 to 1035.

February 3rd.—Had sudden intense lumbar pain on the left side, which passed off in two days. During this month the urine decreased to two and a half and three pints, and he gained seven pounds in weight. The acute pain recurred on February 16th for a day, and there was a trace of blood in the urine on the 28th. During the first half of March his urine again increased to four and five pints, and his weight fell to 103 pounds.

April 12th.—A calculus became impacted in the anterior end of the urethra, and was removed by Mr. Davies-Colley.

17th.—He had severe pain over the lower part of the abdomen with sickness. He could pass his water very freely; poultices were applied; he had little or no sleep. Temp. 100.2°. Treatment: opium, bicarbonate of potash, hyoscyamus; castor-oil enema.

18th.—Sickness abated; pain less; feels weak; no appetite; tongue rather furred; temp. 99.2°. Takes nothing but soda water and a little milk.

19th.—Sickness relieved; much the same in other respects; poultices are being applied. Opium pill; castor-oil enema.

20th.—Tongue rather dry; has been delirious all night; is semi-comatose and still rather delirious this morning. Lies in bed with his legs drawn up and eyes half open. Skin dry and cold. Is semi-comatose. Vomits everything he takes. Temp. 97·3°; resp. 28; pulse 66, thready, compressible, and intermittent. At 5 p.m. he was quite comatose, and died soon after. The kidneys only were examined. They weighed seventeen ounces, and were large, white, and flabby. In the pelvis of the right kidney was a calculus the size of a small pea, and the pelvis itself was inflamed.

CASE 28.—Henry T—, æt. 53. Admitted May 27th, 1874, under Dr. Pavy. He was not severely ill; the urine measured three or four pints, and its sp. gr. was 1030 or 1040. He was dieted. Four days before his death he was taken suddenly ill with pain in the loins. He became drowsy, but could be roused.

The post mortem was made by Dr. Fagge. Emaciation very moderate. There was no distinct odour about the viscera. Cerebral arteries healthy. The brain appeared quite healthy; it was firm. No morbid appearance could be detected in the pons or medulla oblongata. Lungs quite healthy; no trace of pneumonia or phthisis, or even congestion; emitted a fair amount of blood on section. Larynx healthy. Heart 11 oz., healthy. The pulmonary artery contained a considerable quantity of clot; ascending aorta healthy. The intestines showed distinct suction lines, as though there were commencing peritonitis. Stomach much injected, especially towards its cardiac end, which, indeed, was intensely reddened, and in spots ecchymosed. Jejunum and ileum quite healthy, but here and there lined with some opaque mucus. Large intestine dilated, otherwise healthy. Liver 68 oz., of natural colour, certainly not darker than normal; it gave a good reaction of sugar with the copper test. Pancreas normal. Spleen healthy, 6 oz. Kidneys 14 oz.; both were much congested; the pelvis of each was much reddened, and even ecchymosed, and both were somewhat dilated, one more so than the other, and its pyramids were hollowed out. The cortex of this kidney was in a state of suppuration, presenting numerous whitish-yellow points, and the suppuration extending in lines into the pyramids. This kidney also presented some depressed

spots—the cicatrices of a former inflammation—but they were very much more numerous in the opposite kidney, which, on the other hand, had no suppuration. The bladder was much hypertrophied, its walls even a quarter of an inch thick. Its mucous membrane was reddened, and presented some slight white spots, apparently of mucus, easily separable; it contained a little purulent liquid. The urethra and prostate appeared quite healthy; there was certainly no stricture. The mucous membrane at one spot was a little rough; this was in the membranous part. The testes were quite healthy.

CASE 29.—Benjamin B—, æt. 67, was admitted under Mr. Howse's care on June 19th, 1879, suffering from a large carbuncle on the back of the neck. He was found to be passing an excessive quantity of saccharine urine, of sp. gr. 1035. On the 21st the patient was chloroformed, and the carbuncle was opened, and caustic potash and chloride of zinc points were introduced. On the 23rd he was free from pain, but he passed a restless night and was delirious. The following day he appeared better. His bowels, formerly confined, had been opened with magnesia. Temp. 100°; pulse 132, full, regular. June 25th, he became comatose, and died so on the 26th, at 2.30 p.m.

Post mortem.—Body moderately wasted. Brain, lungs, liver, and spleen healthy. The heart weighed 13 ounces. The kidneys weighed 11 ounces; the surface was very granular, the cortex wasted, and opaque and yellowish in colour; the arteries thickened. There was no gout in the great toe-joints.

CASE 30.—Henry S—, æt. 20, under Dr. Pavy's care. First ill in September, 1874, and much relieved by treatment in November and December. Urine very free from sugar in January, 1875. Dieted till August; discontinuing the restriction he had a recurrence of symptoms, and then, in spite of a return to diet, he had cough with blood-streaked sputum. On admission in November the lungs appeared healthy, but on December 17th pneumonia was recognised, and he died January 3rd, having been comatose from the middle of the day before.

Post mortem.—There was diffuse caseous pneumonia of both

lungs, with a large cavity at the right apex. Other organs healthy. (Reported in 'Guy's Hospital Reports,' vol. xxii, 1877, loc. cit.)

Fatal diabetes ; exhaustion (?) ; no post mortem.

CASE 31.—Harriet S—, æt. 30, was admitted under Dr. Pavy's care, October 2nd, 1878. Symptoms of diabetes came on four months ago, and she has had from time to time, in addition to the usual polydipsia and polyuria, cramps and pains in the legs and abdomen. Lately she has had dyspnœa, and slight cough, and has been so weak that she has had difficulty in walking about. Her hair has fallen off a great deal lately. Yesterday she was sick.

On admission she is complaining of severe pains in the abdomen, which is distended and tympanitic. The bowels are constipated. Tongue moist, red, and very sore. Voice hoarse; slight cough; respiration thoracic and abdominal; rhonchi with both respiratory movements; sputa dark, not abundant. Heart normal. Pulse small, feeble. Liver dulness increased. Spleen hard. Urine abundant, sp. gr. 1036, much sugar, no albumen. Menstruation has been irregular, and she had some brownish offensive discharge. The abdominal pain was much worse on October 4th, but better the following day. On October 7th she began the special diet, and the urine, which was 8½ pints daily on admission, with 4500 grains of sugar, had only fallen to 7 pints, with 3700 grains of sugar, on the 13th. On the 15th she took bread against orders, but was altogether better on the 17th. On the 19th she had two attacks of diarrhœa. On the 25th she complained of cramping pains in the abdomen, and of soreness of the throat; this was found to be congested on the right side. October 30th, the throat continued to be sore. She was sleepless, and had pain over the sternum. November 4th, the pain continued. November 6th, she had violent pains in the head. On November 7th she was much worse, with violent pains in the back, chest, and head, and great prostration, and loss of appetite. The following day she was sinking fast, and died on November 9th at 4 p.m. There was no post-mortem examination.

CASE 32.—Alfred B—, æt. 22, admitted under Dr. Moxon on December 13th, 1876. Duration of symptoms three years. Much emaciated, and very weak. The quantity of urine and daily amount of sugar increased until the 19th, and from that date there was a rapid progressive diminution from 10 to 4½ pints of urine, and from 9900 grains to 1000 grains of sugar. On the 20th he complained of severe headache, and on the 23rd was drowsy and extremely feeble. From this date he gradually sank and died on the morning of the 26th; and it is expressly stated in the report that he was not comatose. The brain and spinal cord were the only parts examined post mortem. (Reported in 'Guy's Hospital Reports,' vol. xxii, 1877, loc. cit.)

Diabetes ; phthisis.

CASE 33.—Edward W—, æt. 32, had had symptoms of diabetes four months when he was admitted, emaciated, exhausted, and presenting physical signs of phthisis at both apices. These rapidly became more marked and extensive, and he died a month after admission. There was a little recent pleurisy; both lungs were in a state of caseous pneumonia, and the apices were converted into large ragged cavities. (Reported in 'Guy's Hospital Reports,' vol. xxii, 1877, loc. cit.)

CASE 34.—Walter L—, æt. 21, admitted March, 1880. Diabetes commenced two years and nine months ago. He was partly dieted under Dr. Pavy's care two years ago, but would not adhere strictly to the treatment, and so left the hospital. It was probable, but not certain, that he had phthisis on his second admission. Six weeks later there were obvious physical signs, and he died June 10th, without coma. A large cavity occupied the upper half of the left lung; the presence of tubercle was doubtful.

CASE 35.—E. M—, æt. 19, admitted April 4th, 1879. Symptoms commenced one year and nine months ago. He was treated by dieting in Guy's for some time, but five months ago he returned to ordinary food. Admitted with evidence of phthisis. She died June 5th, and the lungs were found exten-

sively diseased, a large cavity occupying the right upper lobe, with thin walls, and shreddy sloughs adhering. In the left upper lobe were soft rounded nodules of creamy yellow consistence, emitting a creamy puriform fluid on pressure. No tubercles.

CASE 36.—Charles S—, *æt.* 32, admitted in January, 1879. Diabetes first noticed about May, 1877; a year later was under diet treatment at Guy's Hospital, the lungs being then healthy. Shortness of breath was noticed in May, 1879, but the chest appeared healthy as late as June, 1879. In July phthisis became well marked and he died in November. Both lungs were extensively affected with a pneumonic form of phthisis, the upper parts suffering more than the lower. In the cervical region of the spinal cord was an abscess, occupying chiefly the grey matter, and without any indication of yellow tubercle.

CASE 37.—D—, *æt.* 41, had symptoms of diabetes in October, 1878, and was dieted from October, 1874, to February, 1875. His lungs appear to have been healthy in December, 1875, but phthisis was obvious in February, 1876, and he died two months later, with symptoms of pleurisy. There was extensive phthisical disease in patches and clusters of what might have been either yellow tubercle or caseous pneumonia; in some places scattered grey tubercles; and in one upper lobe hepatisation breaking down into a gangrenous pulp.

CASE 38.—George W—, *æt.* 34, had had diabetes two years, and was admitted with signs of phthisis, of which he died a month later. Sugar was absent from his urine shortly before death. He appears to have had no treatment before admission. The right lung was pneumonic from apex to base, but had a large cavity just below the apex, and there were some small cavities at the left base.

CASE 39.—W—, *æt.* 47. The symptoms of diabetes appeared ten months before death, but were preceded for three months by the phthisis of which he ultimately died.

CASE 40.—James C—, *æt.* 45. The disease had a duration

of eight months from commencement to termination. In the third month the lungs were healthy; a month later he had some cough; in the seventh month there was decided slight phthisis, and this became more marked until his death.

The right lung was found extensively cavitated above, and elsewhere tough and yellowish from infiltration with pneumonic products. In the left lung the disease was scattered and nodular. (Reported in 'Guy's Hospital Reports,' vol. xxii, 1877, loc. cit.)

Diabetes ; pneumonia.

CASE 41.—Lucy K—, æt. 25, was admitted on March 1st, 1876. Symptoms of diabetes had existed about a year. She was passing eight pints of urine in twenty-four hours, but under treatment it fell to four or five pints daily. She remained fairly well till May 4th, when she ailed a little and said she had taken cold. On the 6th a pleuritic rub was heard at the base of the right lung; she was very restless and strange in the head, her breathing became more impeded, and she died suddenly the next morning at 7.30 a.m.

Post mortem.—The blood had a peculiar mousy smell. Its colour and coagulability presented no alteration. Both lungs were œdematous, and the right middle lobe was in a state of acute pneumonia; it was not gangrenous. Liver 80 oz., homogeneous in appearance. Kidneys weighed 14 oz. Bladder a little dilated. (Reported in 'Guy's Hospital Reports,' vol. xxii, 1877, loc. cit.)

CASE 42.—Thomas C—, æt. 43. Died with pulmonary symptoms. There was recent pleuritic lymph on the back and sides of the lower lobes of both lungs; cheesy nodules in both apices; no tubercle. At the anterior inferior angle of each lower lobe a patch of grey hepatisation breaking down into minute abscesses.

Diabetes ; syphilis ; suppurative peritonitis.

CASE 43.—Thomas R—, æt. 30. Duration of illness about

twelve months. Had polyuria, with sugar and albumen in the urine, great thirst, œdema of the feet, legs, and scrotum. Was relieved by treatment and diet. On re-admission had peritonitis, and died.

Post mortem.—Acute suppurative peritonitis. Enlarged liver, weighing 108 oz., and containing three small syphilomata. Spleen, 24 oz., lardaceous. Kidneys, 22 oz., mottled yellow, with scattered, depressed marks from local wasting.

OBSERVATIONS
ON THE
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DERMATITIS,
PARTICULARLY
ERYTHEMA, ECZEMA, PSORIASIS, LICHEN,
AND PITYRIASIS RUBRA.
WITH CASES.

By P. H. PYE-SMITH, M.D.

THE inflammatory affections of the skin are by far the most numerous and the most common, and it is here that all systematic writers have found the greatest difficulty. The classification now most generally accepted, that of the late Professor Hebra, was based upon the pathological doctrines of Rokitansky. Accordingly, we find a class EXUDATIONS, which takes up just half of the closely-printed pages of the 'Hautkrankheiten,' and yet does not include lupus, nor ulcers, nor inflammations due to syphilis. The subdivisions of this unwieldy class rest for the most part, not on *pathological* distinctions, but on the *anatomical* basis of Willan's system, while scabies is only separated from eczema by its *etiology*, and pityriasis rubra is defined by its *clinical* features.

I have, in a former volume of these reports,¹ given reasons against this and the many other less successful attempts to classify diseases. These must always be attempts to classify objects which are not homogeneous, sometimes not even definable. If, however, we recognise the different aspects in which "diseases" may be regarded, we may usefully recognise the points in which they agree under each head, whether regarded as anatomical conditions, as physiological processes, as constant clinical combinations of symptoms, as results of antecedent causes, or, lastly, as amenable to curative agents. The first or histological method is useful for definition, the second or pathological for prognosis, the third or clinical for diagnosis, the fourth or ætiological for prevention, and the last or therapeutical for treatment.

Applying this principle to DERMATITIS, we may first draw a marked distinction between the *superficial inflammations*, which affect the Malpighian layer of the epidermis and papillary layer of the cutis only, which never destroy the papillæ, and are therefore never followed by a scar, and the *deep inflammations*, which affect the subpapillary layer of the cutis and the subcutaneous connective tissue, which destroy the papillæ, and always leave cicatrices behind. To the former group belong all the common superficial inflammations of the skin of which eczema is the type, to the latter the deeper and more formidable lesions like lupus and tertiary syphilis. In both groups we distinguish *traumatic* inflammations, due to a known external irritant or injury, from those which are *symptomatic* of a more general pathological process, or which are independent and of unknown cause, *i.e. idiopathic*. Thus, among superficial inflammations, we distinguish Scabies and Eczema solare, and Prurigo pedicularis, as traumatic, from the symptomatic eruptions due to Scarlatina or to Syphilis, and from those which depend on some unknown condition of skin itself. And in like manner we distinguish the deep inflammation caused by a burn from that due to the formation of gummata or of tubercle beneath the papillæ of the cutis, and from the autochthonous processes of Leprosy or of Lupus.

Confining our attention to the more numerous and difficult

¹ Third series, vol. xxii, p. 151.

group of superficial inflammations of the skin, we have no difficulty in separating from the rest so well-marked and peculiar a disease as *Zona*, the inflammations due to Syphilis, the usually slight inflammations caused by the presence of a parasitic fungus, and the special effects due to vermin, *Scabies* and *Prurigo pedicularis*. The remainder, regarded clinically as well as physiologically, appear to fall into the following large divisions :

1. Symptomatic Eruptions, including the *Exanthemata* and *Syphiloderma*. These scarcely need treatment as eruptions, but are of value for recognition of the disease of which they are parts.

2. Allied to these as not being traumatic are the *Erythematous* rashes, which also agree with most of the first class (as measles, scarlatina, enterica) in their anatomical characters, but differ by being not certainly associated with any other morbid conditions. They resemble each other in their slight degree of severity, their fugitive course, their wide and capricious distribution, and their greater frequency among the young.

3. Common superficial dermatitis, *i.e.* such as can be produced in the great majority of skins by an appropriate irritant. Usually traumatic in origin, but probably always combined with a certain vulnerability which responds readily to irritants, which keeps up the dermatitis when the exciting cause has passed, and which sometimes seems sufficient alone to produce a spontaneous or idiopathic dermatitis. This group includes all varieties of *Eczema*.

4. A group of which *Psoriasis* is the type and almost the sole representative, peculiar in histology and in form, incapable of being produced by any known irritant, slow in progress, apt to recur, definite in distribution, unconnected with internal as with external causes, and with its inflammatory character but slightly marked.

5. Lastly, there are the rare, but most interesting, cases of superficial dermatitis, which are universal, which differ from other diseases in their form, which are often accompanied with pyrexia, albuminuria, and other signs of general disturbance, and which not unfrequently end fatally.¹

¹ Of pemphigus, the most important of the idiopathic superficial inflammations

In the following pages I propose to record cases and offer observations on *Erythema* and its allies, on *Eczema* and traumatic dermatitis, on *Psoriasis* and the interesting disease known as *Lichen planus*, and, lastly, on the *Pityriasis rubra* of Hebra and exfoliative dermatitis of Wilson.

I. ERYTHEMA.

If we define erythema anatomically as a superficial dermatitis which does not go beyond the stage of papules, it is impossible to recognise it as a disease. For the term will then include scarlatina and measles, syphilis and enterica, many cases of scabies, and most of prurigo. If, however, we fix our attention on clinical and not only on anatomical features, we shall, I think, admit a natural family of affections of the skin—for the most part obscure in origin, and chiefly important for their resemblance to more serious maladies—which may be fairly called the erythematous group.

We must, however, separate off such slight local dermatitis, set up by external irritants, as intertrigo and the so-called *Erythema leve* of anasarca. These bear the same relation to idiopathic eruptions of the same kind, as *Eczema solare* to true idiopathic Eczema, as pustular inflammation from pediculi capitis to ordinary impetigo of the scalp, or as wheals caused by a stick or a nettle or a caterpillar, to idiopathic urticaria.

Next must be separated cases of abortive or papular eczema, which may be identified by their recurrence, their localisation, and by their being preceded or followed by ordinary "moist tetter."

Thirdly, we exclude erythema where it is merely a concomitant of another primary external lesion, as in prurigo, where the erythema or urticaria so often seen is the result of the patient's scratching.

Fourthly, we put in a separate group erythema which is merely a symptom of an internal primary disease, as in measles.

Is the remaining group of idiopathic non-contagious Erythema natural and homogeneous?

not mentioned in the above list, I hope to offer some observations in a future volume of these Reports.

If we turn to Willan's species of Roseola and Erythema, we shall find no real pathological distinction between them, except in the case of *Erythema nodosum*. Nor do I think that Hebra made a valid distinction between mere hyperæmia (under which he includes several unimportant forms of roseola and erythema) and inflammatory Erythema (*E. exudativum*).

"Roseola," if the term is to be kept at all, should mean a rose-rash without papules, due to whatever cause. Hebra includes two varieties of "Erythema," *E. multiforme* and *E. nodosum*. To these may, as clinical allies, be added *Urticaria*, and two erythematous inflammations of the skin, which go beyond the stage of papules: *Erythema* (or *Herpes*) *iris* and *Erythema bullosum*.

The erythematous group of diseases thus formed agree in the following points:—In their acute or at least subacute course; in not spreading; in frequent return under similar conditions; in causing considerable local irritation; and in leaving no trace behind. They resemble the rashes of measles and early syphilis by a patchy and irregular distribution. They rarely affect the scalp or the flexures of the joints. They are more often seen in children and young adults than in those who have past their prime. The anatomical condition is one of active hyperæmia, often accompanied with acute œdema so as to form wheals, and occasionally producing pimples, vesicles, or blebs. When the congestion is chronic and venous, the œdema may be accompanied with hæmorrhage, as in *Erythema nodosum*.

We know little of the causes of these diseases and less of their rational treatment. They are seldom or never due to an external irritant, but some cases of general erythema and of urticaria are undoubtedly connected with gastric irritation from certain articles of food or from drugs; and this fact makes it probable that other apparently similar cases (especially in children) are also due to slight gastric disturbance. Again, some cases of urticaria, of ordinary erythema (peliosis), and of erythema nodosum, are coincident with attacks of rheumatic fever.

All dermatologists of experience admit the close clinical connection between ordinary erythema and urticaria. I need, therefore, give no cases in illustration of it; but will here

record a few examples of the rarer erythematous affections which are attended with vesicles or blebs.

Vesicular or herpetic erythema.—The outbreak of a little group of clear vesicles on an inflamed patch of skin is not enough to constitute a disease, and there is nothing but an anatomical likeness between the several "species" of Herpes which have been admitted by authors. Willan recognised the distinctive characters of a herpetic eruption and rightly defined it from the anatomical point of view. The species of the genus given by Willan and Bateman are *H. phlyctenodes*, *H. zoster*, *H. circinatus*, *H. labialis*, *H. præputialis*, and *H. iris*. The first of these, preceded by two or three days' fever, irregular in locality, and appearing in successive clusters for nearly the space of a week, may probably refer to zona occurring in other regions than the chest. The third is ringworm of the body. Hebra admits the remaining four species, *H. labialis vel facialis*, *H. præputialis vel progenitalis*, *H. zoster vel zona*, and *H. iris*, including non-parasitic circinate forms. The pathological characters which he gives as common to these varieties are their acute, typical course, their spontaneous involution, and their recurrence at regular intervals. That they are all rapid in their course and curable without interference, is no doubt true, but this is surely not enough to outweigh the marked clinical differences between *H. labialis* and *H. zoster*. Herpes of the face and Herpes of the genitals agree in sometimes recurring, but recurrence of zona is at least as rare as that of scarlatina. Most modern dermatologists therefore separate Zoster (or zona) altogether, in name as in nature, from Herpes. The rare and curious affection called Herpes iris is best grouped with Erythema, as Rayer taught long ago. It may be called *Erythema iris* with Neumann, or simply Iris. One word is better than two, it is distinctive and does not at all events mislead.

The two remaining species of Willan and Bateman may be well united under the name Herpes, the locality affected being indicated by an adjective. This has been done already by Dr. Liveing and other good observers, who have called them febrile, catarrhal, or symptomatic Herpes. Beside the well-known anatomical appearance of the lesion, this disease is characterised

by its acute course, by its spontaneous cure, by its localisation in the neighbourhood of one of the orifices of the body—the nostrils, mouth, urethra or ear—by its not being accompanied by neuralgia, by its frequent recurrence, and by its association with irritation of the cavities near which it appears. I am not aware that it has ever been observed in the neighbourhood of the rectum, but from its painless and rapid course it would be apt, if it did occur here, to be overlooked.

Erythema iris.—CASE.—One of our students, a young man in vigorous health, came to me with a perfect example of the classical “*Herpes iris*” upon the back of his hand. It measured an inch in diameter and consisted of a bulla in the centre, a circle of vesicles, then a well marked injected circle, and lastly, an imperfect vesicular circle at the circumference of the patch. It was unattended with pain or notable irritation, and disappeared in a week. There was no ground for connecting it with indigestion or with any local irritant, and there was no rheumatic history, direct or indirect.

Another case of typical Iris occurred in the practice of Mr. Waren Tay, which he was kind enough to send for me to see. The patient was a boy of thirteen very subject to chilblains. Beside bullous erythema of the feet and ears he had two Iris circles on the back of one hand.

I have now under my care a similar case of *E. bullosum* and iris in a lad of seventeen.¹

E. bullosum is often a mere variety of *E. congestivum* or *pernio*. One sees occasionally cases which resemble a broken chilblain in appearance, but which differ from it by their localization, or their occurrence apart from cold, or their more rapid course.

CASE.—A well-nourished, healthy shop-girl came to me on the 13th of May, 1879, with livid, swollen, pernio-like patches on the fingers, the back of the hands, and the palms. Several had small bullæ upon them. She said that she had never suffered from chilblains of the hands or feet.

I have seen several other cases, both of ordinary chilblain

¹ Dr. Frederick Taylor and Dr. Crocker have lately brought several cases of vesicular and bullous Erythema, Hydros, or Herpes iris before the Clinical Society (Feb. 25, 1881).

and of bullous erythema not apparently connected with cold, affecting the palms of the hands and the soles of the feet. In one child of five years both soles were severely affected, a bubo had formed in the thigh, and it looked at first sight not unlike a bullous syphilide.

CASE.—In June, 1879, a girl of 18, came to me on account of a distressing “flushing” of both cheeks. There was a permanent erythematous patch on the left cheek, livid in colour, with slightly enlarged veins. I was assured that occasionally little bladders formed on such a red patch on the cheek or nose, and exuded a little clear watery liquid. Three weeks later she came again to prove the correctness of the statement, by showing me four or five vesicles on an injected patch on the right cheek. These were as big as split peas, and one had already burst, exuding a drop of transparent yellow serum. The skin was not thickened. There was an erythematous patch on the other cheek also. I could find no connection of this troublesome disorder with the menstrual function, but the patient was subject to flatulent dyspepsia, and also to chilblains (of the feet only) in winter. I advised strict diet and horse exercise, and prescribed alkalies and laxatives, with the local application of collodion. The latter application, she told me afterwards, often stopped it when she was sure it was going to appear by the premonitory flushing and tingling of the face.

A curious erythematous affection has been recorded by Mr. Morrant Baker, the late Dr. Tilbury Fox, Dr. Sangster, and Dr. Cavafy, under the name *Urticaria pigmentosa*.

CASE.—Dr. Goodhart was kind enough to send me a case of this disease from the Evelina hospital. Solomon G—, a child of two years old, has, from the age of three months, been affected with an eruption of rather large, discrete, yellowish-brown papules. They cover the greater part of the back, chest, abdomen, and adjacent parts of the arms and thighs. The hands and feet and the head and neck are free. The rash was not affected by the process of teething. There were two or three fresh wheals of urticaria with erythematous injection around.

I have seen two very similar cases under the care of Drs. Barlow and Sangster. The following case in an adult may, perhaps, be classed as an allied form of Erythema. It also

throws light on the origin of certain forms of Melanodermia and Leucodermia.

CASE.—Eliza B—, æt. 32, lady's maid, of somewhat dark complexion, was sent up to me by Dr. Thos. Fagge, of Ascot, at the end of November, 1880. There was no history of affections of the joints or the skin in the family. She herself had suffered from rheumatic fever at twelve years of age, and had since been liable to palpitation of the heart.

Four years before I saw her, brownish red patches appeared on the abdomen. They itched, though not severely, and they have never entirely left her. Lately she has had a similar rash upon her back. On admission to the hospital she presented a nearly uniform reddish eruption over the back, made up of more or less circular patches, very slightly elevated, smooth, and of a yellowish tinge, which does not disappear on pressure. There are no papules, no scales, and no well-formed wheals. On the abdomen the patches are more separate and gyrate in form, the inside being pale and the edge strongly pigmented. The eruption extends to the flanks, nates, thighs and shoulders, but the head, chest and limbs are free. She says it itches, but there are no scratch marks. I had a watercolour drawing taken by Mr. Hurst, and after other means had been tried, found belladonna liniment successful in relieving the irritation. After eight weeks there was no other improvement. She was soon after attacked with rheumatism, recovered well under salicylate of soda,¹ but had a relapse, which detained her until the end of April, 1881. At that time the redness and irritation had disappeared, but the pigmentation remained.

II. ECZEMA.

Definition and varieties.—Excluding traumatic dermatitis, *i.e.* cases of eczematous eruption in which the lesion corresponds in extent and in duration to the operation of an external irritant, we may perhaps usefully recognise the following as the most important clinical varieties of "eczema," *i.e.* of common, idiopathic,

¹ In this patient, as in many others taking salicylates, I have found the urine reduce copper.

superficial dermatitis, which has reached or will reach the stage of exudation.

1. The most numerous and characteristic group of cases, those which may be called *typical eczema*. The patients may be of either sex and of any age, but are more often young or middle aged adults than children or aged persons. The eruption begins as a papular erythema, but the papules rapidly become small, thin walled, superficial vesicles, which so readily burst under friction that (although almost always present if looked for at the right time) they are in most cases practically absent. A weeping surface thus forms, over which the traces of vesicles may often be discerned (*état ponctué*). As the profuse secretion subsides, thin yellowish crusts appear, the dry surface becomes covered with small dingy scales, the redness and infiltration gradually subside, and the skin returns to its normal condition. Only rarely is accumulation of pigment observed, and never formation of scars. The course of the disease is more or less acute at the outset, but soon becomes chronic, and is apt to return after cure. The distribution is characteristically limited to the thin skin of the flexor surfaces, the favourite places being the bend of the elbows, the hams, and the back of the ears; next, the face, neck, arms and hands, axillæ and groins, abdomen and genitals, thighs and legs; while this form of eczema is rare on the scalp, buttocks and feet. Always more or less symmetrical, it is often as exactly so as psoriasis. Lastly, it is almost always accompanied with itching as well as smarting.

With respect to the pathology of this commonest and most characteristic form of eczema, I confess that I am quite unable to recognise its association with any other disease, or with any supposed diathesis, constitution, dyscrasia, or temperament. It has, I believe, nothing to do either with rheumatism (i.e. multiple synovitis with pyrexia) or with gout (arthritis with deposits of urates of soda), or with scrofula (caseous infiltration of lymph glands), or with rickets, or with anæmia, or with gastric or uterine disturbance. No doubt we see cases of eczema in conjunction with each of these conditions; if this never happened, we should have to investigate the reason of such mutual exclusion; but I believe that it is essentially a disease of the skin and nothing else. As to the blood espe-

cially, we have not, so far as I know, the least reason to suppose that its condition in eczema differs from that of health.

2. *Universal eczema*.—This is a rare form of disease, and many of the supposed cases of it are probably better described as exfoliative dermatitis or pityriasis rubra. But a universal, common superficial dermatitis may occur, which by its localization (when it first appears or after it has become chronic), by its return as a less general affection, or by other characters, claims the title of genuine eczema.

CASE.—Alexander B—, æt. 14, came among my out-patients towards the end of 1877 with inveterate and universal eczema. It was clear that he could not have the necessary attention at home, and I therefore took him into the hospital. He was a thin, miserable lad, of naturally dark complexion; and his whole body had acquired the colour of a mulatto by the gradual increase of pigment. His father and mother and their other children were of ordinary colour and had healthy skins. It appears that he was a healthy baby, but at five years old began to suffer from "scald head." This gradually spread over his body, and though often better and worse has never left him for nine years. On admission, there was dry scaly eczema of the head, face and neck; the ears were fissured and blood-stained, as was the right axilla. The eruption on the arms and back was papular; on the abdomen, genitals, perinæum, nates, and thighs, red and profusely weeping. Except the palms, soles, and part of one shoulder, there was no part of the body free from the disease. The viscera were normal, the urine free from albumen, and the appetite good. Under ordinary local treatment and steadily increased doses of arsenic, in spite of occasional interruption from sickness, the disease rapidly improved, and after five weeks the lad went out with an almost healthy skin. He has several times shown himself again when there has been a slight return of eczema of the scalp or ears, but in other respects he continues well, and has grown into a stout healthy lad; the whole skin continues remarkably dark, but is smooth, soft, and in every other respect normal.

3. *Impetigo*.—The pustular form of common superficial dermatitis as it affects the scalp or face of children is very characteristic and well known. It includes the "scald head," *achor* or

Crusta lactea of older writers, Willan's *Porrigio larvalis* and also *P. favosa*, the *Impetigo larvalis* of Bateman, *Teigne muqueuse*, *Eczema impetigo*.

It rarely affects infants before the scalp is well covered with hair, and still more rarely adults, although we have lately had several cases of typical impetigo of the scalp in both men and women. The exudation is not the characteristic albuminous secretion of eczema, but is purulent, forming thick massive crusts. The eruption is in patches; it is accompanied by comparatively little itching; instead of affecting the ears, limbs, and bend of the joints, it is at first confined to the scalp or face, especially the lips and nose, and when it spreads elsewhere appears to do so by direct inoculation. The three conditions to be distinguished from this true idiopathic pustular dermatitis, are, (1) the pustular inflammation of the occiput caused by pediculi, almost exclusively confined to children, and readily cured by removing the irritant; (2) scabies, which may be little developed on the feet or hands or nates, or may even have been cured and yet has by inoculation of its pus produced "impetigo" of the face or scalp of the child; (3) true ringworm, either obscured by the inflammation it produces, or more often concealed or even supplanted by the severity of the applications which have been used to destroy the fungus.

That impetigo of the scalp and face in children is really a pustular form of eczema is proved, by an ordinary eczema of the scalp or face in an infant becoming pustular and crusted as the patient grows older, by impetigo in a child assuming the characters of common eczema when it recurs, and by impetigo of the scalp being associated in the same patient with vesicular or weeping eczema of other parts. We may connect its peculiarities, first, with the age of the patient. The skin of children appears to be more prone to suppurate than that of adults. Not only eczema but scabies is more often pustular with them; and the scattered pustules of doubtful origin which go by the names of ecthyma and impetigo sparsa are almost confined to children. Secondly, the presence of large and numerous sebaceous glands seems to lead to suppuration, when the surface is inflamed. We know how readily pustules form within the nostrils and in the eyelids (hordeolum), over the shoulders, on the face (acne), and in the beard (sycosis). When eczema spreads to the head in adults

we occasionally see a true impetigo result, but I have never seen pustular eczema on a bald head; here it produces its ordinary clear secretion. Moreover, impetigo is seldom seen on the downy scalp of an infant.

4. *Eczema of the lips*.—This is a curious and somewhat rare form of superficial dermatitis, which used to be called psoriasis labiorum. Its true nature is shown by sero-purulent secretion being present, though in small quantity and forming very thin scabs, and by its association with eczema elsewhere, especially with impetigo of the face. Thus, a little boy with this affection in its most marked form, had also a few pustules on the scalp and ordinary eczema of one ham. Its peculiar appearance is due to the thinness of the skin of the prolabium leading to hæmorrhage, so that the crusts are brown or black and massive. It sometimes affects the lower lip alone. All the cases I have seen have been in children or young girls. The treatment I have found successful is removal of the crusts with bread poultices and application of our Unguentum metal-lorum.¹ The patients are often pale and are benefited by steel.

5. *Eczema rimosum, rubrum, squamosum*, of the hands, including the so-called grocer's and baker's itch, and most cases of what has been called psoriasis palmaris. I have seen two or three cases of true psoriasis of the palms associated with ordinary unmistakable psoriasis, but I believe it never occurs independently. Such cases are either *syphilis squamosa* of the palms and soles, with its small, scanty, dirty scales, its dry surface, its symmetry, and its predilection for the inner side of the sole; or else they are true eczema in its squamous stage.

6. *Eczema of the anus, perinæum, vulva, or male genitals*, extremely irritable, weeping, very rarely pustular.

7. *Eczema of the outside of the forearm and legs*. A form not uncommon in adults, usually rather acute in its onset, appearing in separate round patches, sometimes vesicular, more often presenting the appearance due to broken vesicles, which was described by Devergie as *état ponctué*. Sometimes spreading to the thigh and upper arm, but rarely to the hand or foot; and rather avoiding the usual position of eczema at the elbow and ham.

¹ Containing equal parts of Ung. Zinci, Ung. Plumbi Aret., and Ung. Hydr. Nit.

8. Eczema or dermatitis intertrigo, always weeping and painful, characteristically affecting the folds of the neck in infants, the mammæ in women, the nates, the thigh and scrotum, the groins, and occasionally the toes. I have never seen it on the eyelids or between the fingers.

9. Eczema or dermatitis of one or both legs above the ankle, depending on venous delay, weeping and confluent, œdematous, very chronic, rarely seen before middle age, and often combined with varicose ulcers.

10. The very chronic pruriginous eczema seen in old persons; the lesions usually papular or scaly, with little moisture. To this group belong the worst cases of eczema genitalium and eczema podicis.

11. Chronic, dry, "single-patch" eczema, not itching and lasting for years unaltered. In a patient now under my care, a man of sixty-five, such a patch appeared at forty on the inside of the left thigh, lasted upwards of twenty years without any change, then disappeared, and has for the last eighteen months been succeeded by a similar dry non-irritable patch, as large as a crown piece, on the inside of the right forearm. The old place was called psoriasis, probably incorrectly; this one, which he tells me is just like it, is undoubted eczema, with abortive vesicles. The patient is a hearty man, who has lived freely, but never suffered from gout or anything like gout or rheumatism.

12. *Lichen tropicus*, the acute, intensely irritable, papular, and almost universal "prickly heat" of the tropics. I have lately had two well-marked examples of this curious affection under treatment.

CASE.—The patient is a remarkably tall, well built man of thirty. He was for several years a soldier in the East Indies and was then attacked by this disease. He drank freely, but could not connect the outbreak with a particular debauch nor with any other exciting cause. He has been home some months, and is still much troubled with what is now a very irritable papular eczema. The parts affected are the abdomen, groins, and buttocks; the perinæum and genitals are free. There are also a few scattered papules on the forearms and on both thighs. The irritation continues great, as is testified by numerous scratch marks, but it does not give rise to urticaria. Under

abstinence from drink, free diluents and laxative medicine, he soon improved. The local treatment we found best was an ointment of one drachm of boracic acid made up with equal weights of white wax and lard, softened with oil of almonds. It happened that one of the gentlemen present when this patient appeared had been in Bombay, and another in Australia, and both recognised this affection as the prickly heat with which they were familiar.

CASE.—A soldier, æt. 27, came to me with a papular and pustular eruption distributed over the trunk and limbs; most severe on the abdomen, buttocks, and thighs. The head, face and neck, the hands and feet, and the genitals are quite free. It is very irritable and there are numerous scratch marks. The general aspect is more like prurigo than ordinary eczema. There are some erythematous patches with small wheals from scratching. He says that he had the eruption when he first went to Bengal in 1878. It disappeared in the cold season but returned with the heats. He got rid of it during his voyage home in December, 1880, but it has reappeared this spring.

These are the clinical varieties of eczema which seem to me to be most worth recognising for the practical purposes of diagnosis and treatment. They are varieties, not separate diseases, each of the local kinds being liable to spread into the more common and generalised eruption, and each of the pathological species being liable to assume one of the allied forms. They are all *common* inflammations, *i. e.* such as can be produced at will by an irritant; all *superficial*, not deeper than the papillæ, and therefore, however severe, never followed by scars; all "*moist tellers*," *i. e.* the inflammation is severe enough to cause at one period or other a visible exudation, presenting according to the stage and locality an injected surface, papules, vesicles, raw weeping surfaces, cracks or fissures, pustules, scabs or crusts, a dry, red surface, or branny desquamation. But we never see produced the imbricated scales of psoriasis, nor the large grouped vesicles of zona, nor the rings of tinea, nor the bullæ of pemphigus, nor the large, thin, adherent plaques of exfoliative dermatitis, nor the polymorphic lesions of syphilis. Lastly, all these varieties of eczema are more or less accurately symmetrical, more or less irritable, and run a chronic course, with great liability to relapse.

Ætiology.—On the one hand, we must never lose sight of the fact that eczema is anatomically a “common” superficial dermatitis. For convenience we restrict the name to one which has reached the stage of visible liquid exudation. The exudation may be plasma with but few leucocytes and little fibrinogen, the non-coagulable lymph or “serum” of the older writers, consisting almost entirely of the water, salts, and albumen of the blood: we then have the typical vesicular or weeping eczema; the discharge is abundant, watery, irritating from its saline character, and characteristically stiffening linen from its albumen—*instar seminis*, like white of egg, *comme les sirops*. Or the exudation may be richer in leucocytes than the liquor sanguinis, and these rapidly dying and undergoing fatty degeneration form the opaque, yellowish, milk-like lymph known as pus. But essentially the inflammation is the same, and we see it in all its forms and stages, not only in the various kinds of eczema enumerated above, but also in scabies, which, though rightly distinguished clinically, is nothing but a common dermatitis set up by a special irritant, the eczema of the acarus, or, as Hebra called it, “Scabies eczema.”

On the other hand, we must recognise something besides an irritant, namely, an irritable skin. Often none but the ordinary conditions of the skin as to heat, moisture, cold, and friction can be discovered. Even when we can recognise an exciting cause, as in eczema solare, or in scabies itself, we see that the *quidquid irritabile*, as well as the *quidquid irritans*, is necessary; for two men may be equally exposed to the sun yet only one will be sunburnt; two women may be washing at the same tub, but only one has chapped hands; two children are infested by itch-mites, yet one has only the burrows to show and slight local irritation to complain of, while the other is covered with pustules, scabs, and blisters, over regions which the acarus never visits.¹

Now, what is this *quidquid irritabile* which makes an eczema of a traumatic dermatitis? It is not the normal skin, or we

¹ As in a case of severe bullous scabies, affecting the face as well as the trunk and limbs of a little boy. ‘Guy’s Hospital Reports,’ Series III, Vol. xxii, p. 164, plate I.

should all have eczema from the friction of our clothes and the irritation of soap and water.

It is not "dyscrasia" of the blood or any other "humour," for there is not even a pretence to prove that the blood of eczematous persons differs from that of others.

It is not a poison, an "acridity" circulating in the blood, for why should it affect the skin, and certain parts of the skin only, while the more tender conjunctiva and the more vascular mucous membranes escape? Moreover, in syphilis, where really an infective something is conveyed by the lymph and blood-stream to the skin, the lesions there produced are anything except eczema. When other irritants are carried to the skin—iodides, bromides, belladonna, copaiba, &c.—they produce rashes which simulate, we may almost say which are, acne, erythema, ecthyma, but never what could be mistaken for eczema.

It is not "defective innervation," for if the central nervous system is at fault, why have we no evidence of the brain or spinal cord being affected? If a reflex paresis is set up, what is the seat of the primary irritation? If the fault is in the peripheral nerves, then it is after all a mere local affection of the skin. Besides, we know that in the cases best established of lesions due to injuries of trophic nerves, sloughing of the cornea after division of the fifth, wasting of muscles in amyotrophic lesions of the anterior cornua of the cord, Mr. Hilton's case of ulcer of the finger from pressure on the ulnar nerve,¹ the glossy skin after injury to nerve trunks,² in all these and other more doubtful instances we find either gangrene or atrophy, not eczema, as the result of "defective innervation." But, more important still, we have in zona an affection of the skin which is inflammatory, and which is also clearly connected, by its distribution, by the neuralgia which accompanies or follows or occasionally precedes it, and by direct anatomical post-mortem evidence, with a lesion of the ganglia of cutaneous nerves. Yet zona is not eczema.

It is not a general "constitution" of the body nor a "diathesis" or disposition of the organism; for we see eczema

¹ See Mr. Jacobson's Edition of 'Hilton on Rest and Pain,' 3rd edition, p. 200.

² In cases of gunshot wounds in the American Civil War reported by Surgeon Mitchell.

in persons of all ages, of both sexes, of all races, weak and strong, thin and fat, pale and rosy, dyspeptic and robust, gouty and free from gout.

As to the existence of a herpetic diathesis I shall have to speak presently, when discussing the ætiology of psoriasis. But even granting that there is such a thing, we find no practical agreement among its most eminent supporters as to its limits or its signs. While Prof. Hardy and most of his disciples regard eczema as "l'expression type de l'herpétisme," and only admit in addition lichen, psoriasis, and pityriasis, others, like M. Gigot-Suard, of Cauterets, include under "manifestations primordiales de l'herpétisme" (beside all forms of eczema and impetigo, psoriasis, lichen and pemphigus) acne rosacea, prurigo, urticaria, pityriasis, furunculus, and many more; under *Herpétides muqueuses*, most internal diseases; and as "manifestations ultimes de l'herpétisme," consumption and cancer.

M. Bazin, again, distinguishes between herpétisme (the darts diathesis) and arthritisme (the gouty and rheumatic diathesis), and classifies the varieties of eczema according to their ætiology as traumatic, scrofulous, herpetic or arthritic. "*L'eczème n'existe pas comme entité morbide. C'est une affection générique appartenant à l'ordre des vésicules que l'on retrouve dans plusieurs maladies dont elle ne doit être considérée que comme la manifestation.*"¹ The following are the characters by which herpetic may, according to M. Bazin, be distinguished from arthritic eczema:

Eczéma herpétique.

Spreads.
Limbs chiefly affected.

Symmetry.
Free secretion.
Bright red.
Frequent recurrence.
Itching.
Frequent metastases.

Eczéma arthritique.

Circumscribed patches.
Uncovered parts or mucous orifices.
No symmetry.
Dry, or scanty secretion.
Deep venous red.
Persistent.
Smarting.
No metastases, but previous affections of the joints.

¹ Bazin, 'Examen Critique,' p. 76.

Now, the second column seems to describe the local chronic pruriginous eczema of the anus and vulva, the first common eczema; but I venture to think that itching is more severe in local circumscribed eczema than in ordinary weeping eczema of the face or limbs. As to "metastases" I suppose few pathologists believe in them, but if ever they occur it is rather in the chronic dry eczema of the aged than in the acute moist tetter of the young.

Instead of saying dry eczema means *arthritis*, wet eczema means *dartre*, and pustular eczema means *scrofulide*, when each of these words is so vague and elastic that the assertion is almost as hard to disprove as to prove, surely what facts warrant us in saying is that pustular dermatitis is more frequent on the face and scalp, and profuse secretion on the thin skin of the flexures; that impetigo is commoner with children, and dry chronic eczema with persons past their prime.

When eczema occurs in a thin, pale child, whether with caseous lymph glands or no, we all agree in giving steel and cod-liver oil; when it occurs in a person who has had gout we prescribe colchicum; and when it occurs in a person who bears traces of malaria we add quinine to local treatment, but beyond these limits I do not think that an unprejudiced judgment can at present go. Most cases of eczema are idiopathic, neither traumatic or "diathetic," and we cure them best by local treatment.

Though believing that the true line of progress in dermatology was from Willan to Hebra, and that the fancies of Alibert have been purely mischievous, I do not deny the services of his successors at St. Louis, and especially of M. Hardy. The advance which we owe Bielt in recognising the group of *syphitides* is most important and the aetiological classification which we are now discussing is the ultimate one and the most practically useful of all. But we must follow only proved facts and distrust the guidance of ill-defined terms which are themselves the survivals of systems long proved false.

I admit that the question is much altered in the hands of one who is not a specialist, but a sound pathologist as well as an eminent surgeon. Mr. Hutchinson would associate together as "dartres," diseases which are characterised by relapsing, by symmetry, by chronic and obstinate course, and by distribution

on circumscribed patches rather than diffusely. We are asked to believe that these diseases are due to some unknown constitutional condition which may be called the dartrous diathesis. The diseases so classed by Mr. Hutchinson are psoriasis, pemphigus, many cases of eczema, and a few of lichen, with certain forms of lupus. This list differs from M. Bazin's and from M. Hardy's. If such a clinical group is to be made I should be inclined to add prurigo and pityriasis rubra. But, while recognising certain likenesses between each, each also differs from the rest, and resembles some other affection. I should prefer to admit that all these diseases approach more or less near to psoriasis, but of this I shall presently have to add a few words. With regard to eczema I will only say, that while some chronic dry forms come near to lichen, prurigo, and psoriasis, and some acute and generalised forms approach exfoliative dermatitis and pemphigus foliaceus, the ordinary moist tetter shows rather contrast than likeness to psoriasis, the pustular form differs in almost every point from the "dartres," and some chronic cases of eczema simulate lupus, or elephantiasis, as closely as others do its supposed herpetic allies.

Treatment.—With respect to treatment of the various forms of eczema I have enumerated, the first condition of success is I believe to recognise that the condition is one of ordinary inflammation of the Malpighian and papillary layers of the skin, not "constitutional" or "diathetic," any more than inflammation of the kidneys or of the stomach.

Next, we must look carefully for sources of irritation. It is remarkable that ordinary squalor and neglect produce pustular eruptions, but seldom true eczema; and vermin lead to prurigo or to urticaria, but seldom to eczema. Nor is eczema produced by animal poisons, as are the pustular and erythematous eruptions which we see in the hide workers of Bermondsey, and in butchers. Eczema is the result of the irritation of sweat or of friction, or of exposure to fire, to hot sun, or cold wind. Still more frequently it is produced by the mechanical or chemical irritants used in various trades; as the water in which the washerwoman's hands are kept half wet and half dried, and the coarser kinds of sugar handled by grocers. Eczema of the anus and genitals again may sometimes be

traced to want of scrupulous cleanliness in adults as well as in children.

We must also, in all cases of pustular dermatitis, remember the contagious property of pus, varying greatly in degree, but never to be lost sight of. Many cases of impetigo which seem at first sight to be idiopathic, can be traced, especially in children, to inoculation by the nails from a few pustules produced by scratching an occiput infected with pediculi, or to similar inoculation from a whitlow, from an inflamed phimosis, or from the sores produced by accidental injuries. The spread of scabies itself is not only due to the direct irritation of the acarus and the secondary irritation of the patient's nails, but also to the contagion of the pus. So, again, we see furunculi and ecthyma appear in crops from a single primary source of suppuration.

In most cases, however, and particularly those of the most typical kinds of eczema, we find no traumatic or infective origin. They are strictly idiopathic. Between the purely accidental dermatitis, to which all skins are liable under irritation, and the purely idiopathic eczema where no irritating condition can be found, there is every gradation. As in catarrhal pneumonia, as in dyspepsia, it commonly takes two to make a quarrel. Some persons are exposed to cold yet do not cough, others eat too much and too fast yet suffer no remorse. All I venture to maintain is that the difference between one person and another is not in the "constitution" or "diathesis," but in the anatomical structure (hereditary or otherwise acquired) of the lungs, or of the stomach, or of the skin.

We must then, regarding eczema as dermatitis, treat it like other inflammations, and first and most important is *local* treatment.

We relieve the inflamed skin from the friction of the clothes and as much as possible from that of movement. We protect it from air by rags soaked in lotion, or by smearing it with unguent, or by dusting it with an indifferent powder. And thirdly, we must protect it from water, or rather from the change from moist to dry by evaporation, which is the result of washing.

The late Professor Hebra published not long before his death a characteristic and amusing lecture on the deleterious effects

of water upon the skin.¹ Few of us can be convinced that the daily tub will do healthy English skins anything but good. But there is no doubt that not only soap and water but water alone may be an irritant to an inflamed skin, just as food which is suitable to the stomach in health may be an irritant in gastritis. If we keep an eczematous surface under water, it is soothing so long as the temperature is the same: a continuous bath is sometimes excellent treatment, and there is no objection to the water dressing except from the heat it maintains. But we shall do wisely to forbid washing in the ordinary way in most cases of eczema. Oatmeal, or gruel, or size baths are soothing as well as cleansing if of proper temperature (about 90° F.), and if continued for at least a quarter of an hour; but they are more useful in cases of prurigo, especially infantile prurigo, than in eczema, and should only be used in this disease when the surface affected is large and the secretion free. In eczema, and especially in impetigo of the scalp, the hair must of course be cut short, or in severe cases shaved, the crusts softened with poultices and prevented from re-forming by oil, and the scalp kept clean with equal parts of strained white of egg and water; even this should be sparingly applied and scrupulously dried.

What we want to procure is uniformity of condition. All irritants to living tissues, mechanical, chemical, or what not, are more or less sudden changes. It is possible to heat a frog's muscle until the myosin is coagulated without producing a twitch, or to introduce a constant voltaic current into a nerve, gradually to increase its strength until it much exceeds that of an efficient stimulus, and gradually to diminish it until it can be withdrawn altogether, yet without a negative variation being produced. It is the rapidity of a change, not its amount which acts as an irritant, whether in the normal or the morbid department of physiology.

Poultices or water dressing with gutta percha or india rubber or goldbeater's skin are almost always hurtful from the heat which is produced, whether first applied cold or hot. Nor do I think that we gain by using alkaline water, as used to be the custom at St. Louis. Theoretically, one would recommend "the normal salt solution" of the laboratory with enough

¹ Translated in the 'London Medical Record' for March 15, 1877.

carbonate of soda to make it faintly alkaline. But, practically, it is difficult to prevent even half per cent. alkaline solutions from causing irritation to a raw eczematous surface. Except as prolonged baths or in exceptional circumstances, it is better, I believe, to use moist applications in eczema only as medicated lotions.

Before leaving the subject of water in eczema I may remind the less experienced reader that the cases of general, irritable, weeping eczema are, in old persons, and occasionally in infants, fatal. In these cases continuous luke-warm baths seem to be indicated, but whether really "exhausting" or not, they are supposed to be so, and must therefore be administered with caution. One of my first cases of eczema was in a stout, handsome, healthy old gentleman, with pink skin and silvery hair, whom I saw with Mr. L—. It was widely distributed and excessively irritable. Fresh from Vienna, I ordered a continuous bath, as I had seen it used by Hebra. Great relief followed; but an older and more sagacious physician, who was afterwards called in, while not attempting to cure the eczema, predicted a speedy and fatal result, which soon after happened, and I have not met Mr. L— since.

A short time afterwards, when medical registrar in this hospital, I found, just admitted under Dr. Wilks's care, a patient with extensive and irritable and weeping eczema, also a man above seventy, with clear pink complexion and abundant white hair, and also with a history of gout, though, as in the other case, without evidence of renal disease. I made an unfavorable prognosis of the case. But the patient recovered. I have at the present time an old gentleman of eighty under treatment with extremely obstinate pruriginous eczema, and he appears at present to be equally unlikely to part with his eczema or with his life.

The drugs which we find most useful in controlling local inflammation are those which belong to the group of astringents—zinc, borax, alum, chalk, tannic acid, silver, lead. Of these lead is the most generally and deservedly employed in the treatment of eczema. Zinc and borax appear to have the special additional merit of diminishing irritation. Nitrate of silver is only suited to circumscribed and chronic patches of inflammation.

We have three modes of applying these drugs, as dry powders, in solution, and in suspension or chemical combination as unguents, oleates, or plasters. The general rule I learned from the late Dr. Hughes Bennett is an excellent one: lotions to wet and ointments to dry eruptions. If greasy applications are made to a profusely secreting *Eczema madidans* the discharge washes away the ointment, so that the lead or chalk or zinc never reaches the diseased surface any more than if it were applied over the thick crusts of *Impetigo larvalis*. On the other hand, if lotions are placed in contact with intact epidermis, the horny scales, rendered more water tight by the sebum which covers the surface of the healthy skin, form an almost impenetrable barrier to the action of the drug in solution.

There are, however, exceptions to the rule. Lead lotion is often found to be the best application in the early stages of eczema while still erythematous and in some of its most pruriginous dry forms. Lotions are indicated in hot weather when the skin sweats freely, and ointments in winter when there is no fear of their turning rancid. Lotions are easily applied to infants by the nurse, and to the face and upper extremities by the patient himself; but to be efficient they must be constantly renewed and the surface as lightly covered as possible. Hence they are less applicable to parts of the body which cannot readily be exposed and handled. For the same reason one more often prescribes ointments as a vehicle with hospital out-patients, and those who are about all day, and lotions with in-patients, and those who can or must lie up at home and devote themselves to their cure. Lastly, we meet with certain cases in which our patients assure us that either lotions or ointments always disagree with them, and I have too often verified this assertion to neglect it.

When there is much clear serous effusion, and especially in eczema of the folds of the limbs, powders are often better than either lotions or unguents. Finely powdered chalk, oxide of zinc, or zinc and starch, dry up such weeping surfaces and form a false scab under which the healing process goes rapidly on. The intertrigo of infants and of the breasts in women is often quickly relieved by first cleansing with white of egg, carefully drying, and then powdering with oxide of zinc. This plan is,

however, ill adapted when the affected parts are allowed to be in motion, as with intertrigo of the nates. In such cases the ordinary benzoated zinc ointment, with double the official quantity of zinc oxide so as to make it drier and firmer, or vaseline with zinc, are better applications: they should be spread upon thin rags and kept in place with a suspensory bandage between the thighs. When a similar condition arises from riding or rowing, the interval between the periods of irritation are longer, and it is possible, by scrupulous cleanliness and diligent powdering, to procure healing without altogether stopping the cause.

There are, however, some cases of eczema, especially, I think, in the young, and of the moister kinds, which resent every kind of medication, and can only be treated by the sedulous employment of the most soothing and indifferent applications. Among these, as I have already pointed out, water cannot be reckoned; it almost always does harm, and alkaline washes are worse. Thin size, cold cream or vaseline, I think, are the most likely to succeed in such cases. Glycerine of starch occasionally succeeds when everything else seems to fail, but it often proves extremely irritating, and on the whole is I believe less used than it was some years ago. Glycerine has the advantage of mixing freely with water and may thus be used as a vehicle of tannin or of borax to moist surfaces and mucous membranes; but the withdrawal of water from the surface appears in itself to be sometimes an irritant.

I am convinced that for the common "eczema solare" of Switzerland and of the sea glycerine is with most persons of little or no use, and is far better replaced by vaseline, which should be gently rubbed in before the face begins to sweat, and renewed from time to time while the exposure continues.

After the acute stage of an eczema has passed, and it is as a chronic inflammation that it usually comes before us, astringents are still indicated, but instead of soothing and protection some stimulation is necessary. This we obtain by adding a mercurial salt, and the *Ung. Metallorum* of our Guy's Pharmacopœia is one of the best combinations for treating impetigo and chronic eczema. The red oxide ointment, either alone or added to *Ung. Zinci*, is also very valuable, especially in the most chronic and indolent forms which approach ulceration.

In obstinate eczema, especially where of small extent and moist, the application of *Liquor Potassæ*, as advised by Dr. McCall Anderson, is often an efficient and rapid means of cure. Acid nitrate of mercury is the best application for local deep fissures of inveterate eczema *rimosum*. In the very chronic eczema, which is dry and scaly without much active inflammation and itching, tar ointment, or our *Ung. Liq. Carbonis detergens* is indicated. For single patch eczema, if wet, *Liq. Potassæ*, if dry, *Ung. Picis liqidæ* is best.

Meantime we must prevent the irritation of scratching and rubbing by relieving its cause. Oxide of zinc, calamine, or borax, as ointments, weak corrosive sublimate wash or *Ung. Hydrargyri ammoniati*, hydrocyanic acid lotion (ʒiv—ʒvj to a pint), cyanide of potassium ointment (gr. ij to the ounce), and mere protection from the air by such indifferent applications as cold cream and vaseline :—these are all useful for the purpose indicated, the poisonous sedative being used with caution when the moist surface is extensive. One advantage of the continuous bath is the relief from itching it affords. But beside external remedies it is important, especially with children, to add sedatives to secure rest for the inflamed skin at night. Opiates are undesirable not only on general grounds for children, but also because they occasionally aggravate pruritus. Henbane is better, and combined with camphor as a pill or a draught often seems to suit old men better than any other hypnotic. Chloral hydrate is particularly adapted to children, and is best given to infants alone. With older children and adults, bromide of potassium and chloral hydrate make the best combination.

In obstinate cases of eczema of the hands the following method is almost always successful. Wash them thoroughly, removing all crusts, secretion, and dead epidermis, and cutting the nails short. Then rub vaseline gently in all over, put on a well fitting pair of kid gloves and keep them on night and day, only removing them for applying fresh vaseline.

But with all our care we find local remedies inadequate to the cure of perhaps half our cases of eczema. Even at Vienna medicines are taken internally in this disease.

Impetigo rarely needs physic, but when the child is pale steel certainly hastens the cure, beside doing good otherwise. Steel wine for infants, the saccharine carbonate of iron for older

children, and the citrate of iron and quinine are all useful in the treatment of children's eczema ; but when they fail, and steel is still indicated, it is worth giving the tincture of the perchloride in glycerine and water before trying arsenic. In the severe form of chronic eczema in children, especially when not punctular and when widely distributed, arsenic is almost always necessary, and rarely fails of success. It must, of course, be begun in small doses, and must always be given with food, and then it scarcely ever disagrees. Children bear it very well even in full doses, and grow fat and rosy while taking it. I have given as much as fifteen drops of Fowler's solution three times a day to a child of seven years old without any but good effects. Sometimes the soda agrees better than the potash salt and it should always be well diluted with water. Except a little syrup to sweeten it no adjuvant is needed. Perseverance in this treatment is rewarded in the most inveterate cases. I have given the above in a case of universal eczema which existed from infancy till near puberty in a boy, and was cured at last in a few weeks by sedulous local treatment and persistent exhibition of arsenic notwithstanding sickness. I had equal success with a girl of sixteen, who had been subject to eczema from early childhood, and suffered terribly from its excessive irritability and its deformity. She could never go out without a thick veil, and every evening was obliged to retire to her bedroom on account of the itching, which then became intolerable. Under arsenical treatment the disease completely disappeared, and she is now able to go into society. This case was one of those which were once explained by the theory of "metastasis," for when the eczema was least troublesome the patient was subject to asthma, and this disappeared in spring and autumn, when the eczema became most severe. Since, however, the skin has become normal the cough and dyspnoea have also improved.

In some children with more or less obvious signs of tubercle in the lymph glands or elsewhere, the exhibition of cod-liver oil certainly appears to hasten the cure of eczema, as well as to improve their general health.

I have already referred to the importance of hypnotics in the treatment of irritable eczema in children.

In the acute weeping eczema of adults experience confirms

the practice of giving saline laxatives along with local treatment. Friedrichshall or Carlsbad water, Epsom salts, and the "white mixture" of magnesia and sulphate of magnesia, or carbonate of soda with sulphate of soda and sulphate of magnesia, are the best kinds of purgatives. Occasionally soda and rhubarb succeed better. In the eczema of children few internal remedies are so useful as Gregory's powder. The value of mercury internally administered is almost confined to the disease in young children, and in some cases we cannot doubt its benefit. The carbonate of soda three parts with Hyd. c. Cretâ one part of our pharmacopœia is the best form in which to give it.

Iron is seldom needed unless obvious anæmia is present. But with women, especially towards the menopause, sulphate of iron with sulphate of magnesia forms an excellent combination, especially if a few drops of dilute sulphuric acid be added.

In the irritable and obstinate eczema of elderly persons arsenic often appears to aggravate the malady. Local treatment assisted by internal sedatives is in these cases most useful, but occasionally purges appear to be of service, and certainly add to the physical and mental comfort of the patient. I believe that eczema occurs more often in connection with gout than does psoriasis or any other disease of the skin. In such cases colchicum is undoubtedly indicated.

The local persistent forms of eczema rarely benefit except by careful and persevering local treatment; but in the ordinary chronic dermatitis of the legs in elderly persons laxatives are no doubt a valuable adjunct to elevation of the limb, and artificial support of the enfeebled veins by flannel rollers, elastic stockings, or, best of all, by Martin's india-rubber bandage.

As to diet, we are for the most part content to follow the traditional warnings against salted food, spices, and preserves. I am sure that most children suffering from eczema benefit by a meat diet and some of them by the addition of stimulants. This also applies to anæmic adults, and especially to the case of women suffering from over-lactation or from menorrhagia. On the other hand, adults in general appear to benefit by taking less meat, no malt liquors, abundant diluents, and plenty of fruit and vegetables. In cases of the pruriginous

eczema of the aged abstinence from fermented liquors is sometimes successful, though I have more than once known it fail.

The regulation of the diet of infants suffering from eczema is of paramount importance. We often find that before they are fully weaned infants are fed upon potatoes and other food containing indigestible cellulose or excess of starch. It is easy to see the mischief of this. But even when milk alone is given it often causes irritation, as shown by diarrhoea and vomiting; and if our local remedies are to succeed we must dilute it or mix it with lime water. Infants suffering from dermatitis, with consequent pyrexia, often "crave" for the breast or the bottle, not from hunger, but from thirst, and thus complete a vicious circle by overloading their stomachs with food, when, if they could express their wants, they would ask for water.

On the whole, internal treatment is most likely to be of value when used to help careful and energetical local treatment.

III. PSORIASIS.

Frequency.—Next to eczema, scabies, and syphilitic eruptions, psoriasis is the commonest disease of the skin among London out-patients.

Of 180 consecutive cases noted for the purpose in January and February, 1879, I found the numbers to be of eczema 45, scabies 29, psoriasis 19, syphilis 18, impetigo capitis 11; of 179 consecutive cases in the same months this year (1880) there were of eczema 32, scabies 27, psoriasis 16, syphilis 10, and impetigo capitis 32. In three summer months (June, July, and August) of 266 consecutive cases 37 were impetigo, 59 other forms of eczema, 23 scabies, 21 syphilis, and 18 psoriasis. Uniting the three lists the proportion is of eczema 22 per cent.; of scabies, 12·5; of impetigo, 13; of psoriasis, 8·5; and of syphilis, 8.

Comparing these figures with those of other observers we find that of the enormous total of 10,000 consecutive cases observed in hospital practice in Glasgow by Dr. McCall Anderson, 2527 were eczema, exactly the same number scabies, 725

psoriasis, 567 ringworm and other *tineæ*, 517 syphilodermia, and 327 phthiriasis. The same physician found among 1000 consecutive cases in private practice 348 of eczema, 106 of psoriasis, 101 of erythema, 57 of syphilis, 86 of ringworm (beside four of favus, which is less rare in Scotland than elsewhere), 44 of scabies, and 54 of acne, and 21 of rosacea (i.e. gutta rosea or acne rosacea).

Of Mr. E. Wilson's 1000 consecutive cases observed in private practice, 298 were eczema, 112 acne (or gutta) rosacea, 78 psoriasis (alphos), 55 acne, 39 ringworm, 37 scabies, and 30 syphilis.

At the Bellevue Hospital of New York, Dr. Bulkley found among 1000 consecutive cases, 302 of eczema, 111 of acne, 98 of syphilis, 57 of phthiriasis, 50 of psoriasis, 48 of *tineæ*, and 36 of scabies.

Of 11,000 cases collected by the American Dermatological Association, from private and hospital practice throughout the States, more than 8000 were eczema, 1414 syphilis, 685 acne (excluding gutta rosea); and next came psoriasis with 402 cases, followed by ringworm with 356, and urticaria with 333 ('Tr. Amer. Derm. Assoc. Philadelphia,' 1881).

Terminology.—The name psoriasis is, like most others in dermatology, of purely conventional significance; it is not a "condition of psora," for it has nothing to do with scabies, and in most cases is attended with less itching than prurigo or than chronic eczema. But the name is distinctive and universally recognised, so that there is fortunately no chance of "alphos" or any other displacing it.¹ Happily also the artificial and misleading use of lepra as a synonym of certain supposed forms of psoriasis is now almost forgotten. One can only wonder that such an acute observer as Willan should have admitted the distinction between lepra and psoriasis against the evidence of his senses, in order to follow the confused and sometimes misinterpreted descriptions of Greek and Latin authors.

Hippocrates ('Aphor.,' iii, 20) speaks of *leprae* together with

¹ "Pour citer un exemple, le psoriasis d'Erasmus Wilson n'a rien de commun avec l'affection ainsi nommée en France," writes Dr. Vérité, and quotes from Mr. Wilson: "Psoriasis is a mitigated and chronic form of psora or ekzema."

lichen and *alphosae* diseases which occur in the spring of the year. Galen ('De tumoribus,' xiii) makes *psora* and *lepra* "melancholic diseases of the skin alone; if they affect the veins and flesh they are called *cancer*." Paulus Aegineta (lib. v, cap. 89) also puts *psora* and *lepra* together, as roughness and itching of the skin, "proceeding from black bile," i.e. melancholic; but he distinguishes *lepra* thus: διὰ βάθους ἐπινέμεται τὸ δέρμα κυκλοτέρως, μετὰ τοῦ φολιδοειδῆς ἀφεῖναι λεπίδας. This does not so well apply to psoriasis but rather to the squamous and ulcerative stages of cutaneous syphilis. Actuarius (lib. ii, cap. 20) describes *lepra* as less formidable than *elephantiasis* (a term not used before Celsus and Aretæus), the next in severity being *psora* (scabies), and then *lichenes* (impetigines). *Lepra* goes deeper than the latter and wastes away the skin (τίνας συνήξει σὰρκος ποιεῖ) and gives off scales—a mere repetition of the statement of Aegineta.¹ Herodotus speaks of persons suffering from *λέπρα* or *λέγκη* being compelled to live separately in Persia (i, 138). The terms here are no doubt synonymous with what would still be called scaly and white leprosy respectively.

The Septuagint translators used *λέπρα* as the equivalent of the Hebrew זאַרַּתַּח, and *λεπρός* is the word for a leper in the New Testament. Thence the word passed into all European languages, with the adjective *leprosus*, from which our form 'leprosy' is derived.

After the word *elephantiasis*² was introduced, it was supposed to denote the most malignant kind of leprosy. Thus in the passage quoted above, Actuarius says: "*Lepra* is a less evil than *elephas*; after it again comes *psora*, and then *lichenes*."

After the revival of learning, Gregory Horst, of Nuremberg, in his 'Epistola de Hymene et Lepra' (17—), distinguishes *Elephantiasis Arabum* as "a disease of the feet with great swelling and distended veins," and correctly describes the

¹ This and the two preceding citations I came upon in the exposition of Greek words used by Hippocrates, Aretæus, and other medical writers, published by Henry Stephen, in 1664.

² "*Elephantiasis Græcorum*" is a clumsy expression, which has produced endless confusion of leprosy with Pachydermia. "*Leprosy*" and "*Barbadoes leg*" have each a definite meaning, *Elephantiasis* has none.

elephantiasis of the Greeks as the same with the lepra of the Latins and Arabians ; i.e. as leprosy (*Aussatz*) ; and he describes the lazaretto houses of Germany at that time.

In the eighteenth century the learned Dr. Mead, in his 'Medica Sacra,' heads the chapter on the leprosy of the Bible with the title *Lepra morbus*. He says that lepra is a kind of scabies, and speaks of elephantiasis as *leprae congener morbus*. After quoting the well-known accounts of Celsus and Aretæus he concludes : "*Ex his igitur omnibus manifestum fit, lepram in Syriâ non naturâ sed gradu tantum ab illâ in Græciâ quæ λεύκη ibi vocabatur diversam fuisse ; et ipsum hunc morbum interdum apud Græcos, maximè verò inter Arabas elephantie affinem fuisse.*"

Dr. Daniel Turner, in his 'Treatise of Diseases incident to the Skin,' 1723, describes Barbadoes leg as leprosy of the Arabians, and elephantiasis as leprosy of the Greeks.

Blancard's 'Lexicon Medicum' (Lugd. Batav. 1702), which correctly distinguishes Elephantiasis Arabum, *de quo morbo ne verbum quidem fecerunt Græci*, from Elephantiasis Græcorum *quam Arabes lepram vocant*, describes true leprosy as "Elephantiasis, sive lepra et leprosis ;" and translates it "*Aussatz, Lèpre, the Leprosie.*"

Heberden and Cullen both affirm that they had never seen lepra, but the former describes psoriasis clearly enough as "a branny scurf observed in patches all over the body, and very apt to begin at the point of the elbow." So that the difficulty of which Bateman speaks¹ arises only from his refusing to recognise lepra as a term for leprosy.

Diagnosis.—The discrimination of psoriasis from scaly syphilis is occasionally difficult, but in most cases the large, glistening scales, the colour, the characteristic distribution, the uniformity of lesion, the irritability, and the recurrence of the attacks in precisely the same form, distinguish the former from the latter disease, apart from the absence of other signs of syphilis.

¹ "It is difficult, therefore, to account for the opinion expressed by the late Dr. Heberden respecting the extreme rarity of Lepra in this country. And still more difficult to explain the statement of Dr. Cullen . . . that he had never seen the disease,"—'Practical Synopsis,' p. 28, note (ed. 1824).

Most of the cases which were formerly described as *psoriasis palmaris* were no doubt squamous syphilides, and others seem to have been chronic *Eczema rimosum*; but I have certainly seen true psoriasis affecting the palm, once in Vienna and twice at least in my own practice. In each of these cases the occurrence of psoriasis in the usual situations made its recognition easy. The distinction of true psoriasis from eczema squamosum (i.e. of alphas from psoriasis, according to Mr. Wilson) is easy enough in practice; the confusion is only one of words. The distribution, the size and colour of the scales, and the previous condition of the skin, are amply sufficient to distinguish them.

Etiology.—With respect to the origin of psoriasis, I am entirely incredulous of its connection with gout or with scrofula, or with any imaginary diathesis, dyscrasia or temperament. It is a disease of the skin and nothing else. I do not deny that psoriasis may occur in a patient who has urate of soda in his joints, or in a child who has caseous cervical lymph glands. If we never met with such cases, it would follow that gout or scrofula protected from psoriasis. But one may certainly see marked and inveterate psoriasis in the most varied conditions of health, in the most robust and ruddy, as often as in the thin and pale. It is not a disease of the blood, nor of the humours, nor of the nerves, but of the skin; and is as independent of other lesions as any other histologically local disease.

Bazin, Hardy, and French pathologists generally, supported by some authorities in this country, have assigned to psoriasis a leading place in the group of dartrous diseases, which has already been criticised under eczema. The hypothesis of a dartrous diathesis and the entire order of ideas to which it belongs, appear to me to be baseless in fact, unscientific in principle, and useless or harmful in practice.

Willan wisely discarded the imaginary *virus dartreux*, which was in fact nothing else than the psoric humour of Hahnemann (*la gale partout*) a humour now (I believe) given up even by his own followers. The disciples of the English school of dermatology in France—Biétt, Cazenave and Gibert, and Devergie maintained the same scientific and practical attitude, not framing hypotheses but observing facts. But the dartres were

again brought into notoriety by the presumptuous and hasty dogmatism of Alibert, and have since, under various modifications, been recognised by his successors at St. Louis, by Bazin, Hardy, Caillaut, Guibout, and many others.

If any one wishes to judge of the lengths to which this doctrine has been carried, I would recommend the perusal of the bulky volume on "*Herpétisme*" by M. Gigot-Suard.

Taking with all respect the statements of the eminent French physician, M. Hardy, we find that the characters of the darts are: 1, They are not contagious; 2, they are often hereditary; 3, they recur; 4, they itch; 5, they spread; 6, they are chronic; and 7, they do not leave scars.¹ By these characters "we are led logically to believe" that these darts are due to *un vice dartreux*, *virus dartreux*, or, as M. Hardy prefers to call it, *diathèse dartreuse*. The diseases, which are not local, but true darts, are beside psoriasis, eczema, including impetigo, lichen, and pityriasis, including pityriasis rubra. To these other authorities add pemphigus, erythema, and many other kinds of disease, so that, excepting syphilis, herpes and cancer, few affections of the skin have not been brought more or less under the comprehensive dartrous hypothesis.

We are told that it is possible to distinguish a dartrous diathesis apart from its manifestation in actual disease of the skin. "*Les personnes dartreuses, bien qu'ayant en apparence tous les attributs de la bonne santé, sont cependant dans un état particulier qui n'est pas la santé parfaite.*" Their skin is dry, and they do not easily sweat. Their skin readily itches, and is inflamed by slight causes, such as eating shell-fish (which makes *urticaria ab ingestis* a "dartre," contrary to M. Hardy's classification elsewhere), and they have a good appetite even when they are ill. These characters (excepting the last) appear to me to apply to eczema and to eczema only. When the skin is irritable and readily inflamed we may say, if we please, that it is "disposed" to common superficial dermatitis, i.e. to eczema;

¹ "Nous appellerons *dartres* des affections de la peau à lésions élémentaires différentes, non contagieuses, se transmettant souvent par voie d'hérédité, se reproduisant d'une manière presque constante, présentant pour symptôme principal des démangeaisons, toujours disposées à envahir de nouvelles régions, à marche habituellement chronique, et dont la guérison a lieu sans cicatrices, bien qu'elles s'accompagnent souvent d'ulcérations."—*Leçons sur les Maladies de la Peau*, p. 19.

or that the skin (not the person) is of an eczematous construction or "constitution," of an eczematous disposition or "diathesis." But patients who often suffer from eczema are not particularly liable to psoriasis, nor to acute erythema; and there is no reason to suppose that their blood (any more than their nerves or brains) is different from other people's.

Looking to the signs given above, which are to justify one in assigning an actual eruption on the skin to a dartrous origin, we find none of them sufficient. (1) Lupus is not contagious yet it is not a dartre; pustular eczema is often contagious yet it is a dartre. (2) Hereditary transmission proves only that a disease is not accidental or traumatic, not contagious and not parasitic. What is hereditary is a certain structure of the skin, as of other organs, as stature, as malformations, as shape of limbs and head and nails, as colour of skin and hair and eyes; or again, functional peculiarities, as early or late baldness and greyness, early or late atheroma. Every one admits that eczema is often hereditary and that psoriasis is so also; but what wants proof is that any common state is transmitted which may turn to one or the other. Moreover, other diseases of the skin not considered dartrous are often hereditary, as cancer, leprosy, and even erythema nodosum. (4) Itching is less characteristic of psoriasis than of prurigo, urticaria, and scabies. (7) Not leaving scars shows only that the papillæ are not destroyed and applies to all other superficial affections of the skin. The remaining characters, chronic course, gradual spreading, and aptness to recur, are no doubt points of agreement between eczema and psoriasis.

That there is a pathological relationship between them may be admitted, but it is chiefly one of contrast. Eczema is often acute or subacute, psoriasis is chronic; eczema is at one time or other moist, psoriasis never; eczema affects the flexures and the thinnest parts of the skin, psoriasis the most exposed and thickest regions: eczema is the most varied of diseases in its outward form, psoriasis the most constant; eczema can be and often is produced by direct irritants, psoriasis is always idiopathic.

Apart from the special question of the origin and nature of psoriasis, I may here be allowed to repeat that the whole order of notions expressed by such terms as "constitution," "dia-

thesis," "temperament," appears to me to be a survival of exploded physiological systems and only obstructive of investigation. Constitution means a certain structure of the solids of the body. Temperament means a certain tempering or mixture of the humours of the body;¹ dyscrasia an ill mixing of the same humours. But the humoral pathology is dead. There is no mixture of blood, bile, phlegm, and black bile. The spleen is not a gland, and melancholic patients are not, by the testimony of the deadhouse, "splenetick." If there was any accidental truth in the doctrines of the humoral pathology, it must be proved anew by careful investigations into the prevalence of certain diseases in the lower races of mankind. Certainly so mixed a population as that of England is ill fitted for such inquiries.

It was once believed that the skin was a chart on which the humours of the body displayed their signs for the scrutiny of the physician, just as we now look on the tongue chiefly as an index to the state of the stomach. There was foundation for such a theory in the case of jaundice, of syphilodermia and of febrile rashes. But we now know that most affections of the skin are strictly local and structural. If it can be proved that psoriasis occurs more often than the doctrine of chances would explain in persons subject to gout or to scrofula, and if we thereby learn how better to treat our patients, the proof will be a welcome addition to science and to practical therapeutics. But gout must mean not an arbitrary assumption, but the existence of urate of soda in the tissues; and scrofula

¹ "If the Element of Fire be Chieftain, the Body is said to be Choleric; if Air bear rule, to be Sanguine; if Water be in his Vigour, the Body is said to be Phlegmatick; if Earth have his Dominion, to be Melancholick. For Choler is hot and dry, Bloud, hot and moist, Water, cold and moist, Earth, cold and dry. These four complexions (or temperaments) are compared to the four Elements, secondly to the four Planets, Mars, Jupiter, Saturn, Luna, then to the four Winds, then to the four Seasons of the year, fifthly unto the twelve Zodiacal Signs, in whom are four Triplicities, lastly to the four Ages of Man; all of which are here deciphered and limned out in their proper orbs, thus: I. *Choleric*, Aries, Leo, Sagittarius; Mars, Ignis, Favonius, Aestas, Juventus. II. *Sanguine*, Gemini, Libra, Aquarius; Jupiter, Aer, Auster, Ver, Adolescentia. III. *Phlegmatick*, Taurus, Virgo, Capricornus; Luna, Aqua, Autumnus, Vergens, Aetas. IV. *Melancholick*, Cancer, Scorpio, Pisces; Saturnus, Terra, Aquilo, Hyems, Senectus."—*The Optick Glasse of Humours*, 1664.

must not mean vaguely ill-health, but caseous degeneration of lymph glands.

Until, therefore, we learn better, we must consider that psoriasis is not due to a supposed arthritic, herpetic, or dartsious diathesis, that it is not a manifestation of gout, and that it has no more to do with scrofula than with syphilis.

Again, we cannot trace psoriasis, as we so often can eczema, to a local irritant. It cannot be excited at will, it is not produced by sun or cold, or sweat or friction, or mustard, or venous congestion. It is scarcely an inflammation, certainly not an exudation in the sense of Rokitansky and Hughes Bennett.

But the histological appearances are decisive of its being a true chronic inflammation of the Malpighian layer and sub-jacent papillæ, with hypertrophy of the latter and subsequent atrophy of the former.¹ And occasionally we see the early stages of psoriasis, like those of syphiloderma, showing the ordinary signs of inflammation.

CASE.—A young man came to me with a bright rose rash, which had appeared the day before in minute patches over his chest. It looked like early syphilitic roseola, but there were no other secondary symptoms and no evidence of infection. There was slight local heat and general malaise. Two days later he came again, perfectly well in himself, with the rash changed into a papular form and several of the papules covered with small white scales. In a week or ten days an ordinary *Psoriasis guttata* had developed, which soon yielded to remedies and has not returned.

The early origin of psoriasis, its almost constant distribution, its frequent repetitions, and its appearance in different members of the same family, point to its being an inherent and not an accidental vice of the skin.

Though most cases of psoriasis appear to be free from hereditary influence, we sometimes meet with such marked instances that we must admit it among hereditary diseases. In this respect it resembles carcinoma and rheumatism.

I need scarcely refer to a lately propounded hypothesis that psoriasis depends upon the presence of a fungus, for it is

¹ See the figures by Neumann, and an interesting paper by Dr. Thin, in the 'British Medical Journal' for July, 1881.

contradicted by all we know of its course and origin, as well as by microscopical investigation of the scales and of the skin in section. Dr. Yandell, of St. Louis, recognised several of my cases as malarial in origin, but the evidence did not convince me.

Form and distribution.—The form of psoriasis is remarkably constant, and its distribution scarcely less so. It is always the dry, scaly tetter, always bilateral, and often exactly symmetrical, but never, I believe, universal. Symmetry has been said to prove that a disease is "constitutional." If this means only that it is not traumatic, it is true enough, but if it means that the symmetrical disease is due to some anomaly of the blood like leuchæmia, or to some generalised condition like carcinoma, I can see no justification for the dictum. The lesions of the skin in purpura, an affection of the blood and blood-vessels, and in syphilis, a generalised disease, are far less symmetrical than in psoriasis and idiopathic eczema, which are both strictly confined to a single organ. The two elbows are not covered with scales because they are both supplied with the same blood, for no part of the skin (or mucous membrane either) has its private supply of nourishment. The two elbows are affected with psoriasis because their skin is more alike than that of any other part of the body. Next in likeness is the skin over the knee caps, and least so the skin of the axillæ.

The varieties described as *Psoriasis punctata, guttata, nummulata, annulata, gyrata*, &c., depend chiefly on the period at which the disease is observed, and are of no scientific, that is, of no practical importance. There is, however, one form of this affection which seems to me to be distinct enough in more than mere accidents of appearance to deserve notice as a variety.¹ (1) It is guttate in figure, the separate spots not coalescing as usual into larger patches; (2) there is little redness around the scales; (3) the distribution is much less regular than usual, the whole trunk being often spotted over and the elbows and knees free; (4) it scarcely itches at all; (5)

¹ Since writing the above I have noticed in Dr. Liveing's excellent 'Handbook of the Diagnosis of Skin Diseases,' p. 120, a very similar account of what he called Scrofulous Psoriasis.

it occurs almost invariably in children; (6) it often does not require arsenic, but is successfully treated with cod-liver oil, or sometimes with steel.

This want of conformity to the typical geography of the disease is what may be observed in other cases of children's pathology, in pneumonia, for instance, and in tubercular disease of the abdomen, in affections of the joints, and of the eye. In the case of the skin we may perhaps say that the distribution of morbid processes is less precise than in the adult, because the several regions of the skin are less differentiated, because the skin of the elbow, the hand, the back, the chin, is much more alike in the child than in the man.

Psoriasis of the nails and of the tongue.—The nails are seldom affected except in the most severe and extensive forms of the disease; yet malformations of the nails are more frequently due to psoriasis than to eczema, ringworm, syphilis, or any other general disease of the skin.

Beside the well-known patches of thickened epidermis on the dorsum of the tongue, which go with fissures, nodes, and other undoubted syphilitic lesions of the part, and beside the chronic indolent patches of "psoriasis" or *ichthyosis linguae* which precede epithelial cancer, there is a true psoriasis of the organ, which though rare may be met with in cases of the disease; and I have twice observed it in connection with lichen planus of the body.

The following are notes of the points indicated in fifty-five consecutive cases of psoriasis lately under treatment:

No.	Sex.	Age.	Form and distribution.	Duration.	Family history, &c.
1	M	35	Ordinary <i>Ps. diffusa</i> ; elbows and knees affected	33 years; once free for 3 years	One brother.
2	F	18	Ditto	Since childhood	
3	M	Adult	Ditto	6 months.	
4	F	9	Ditto	2 years; every spring and fall.	
7	M	7	Ditto, extensive; face, scalp, hands, and feet only free	2 years.	

No.	Sex.	Age.	Form and distribution.	Duration.	Family history, &c.
8	M	22	<i>Ps. guttata</i> ; elbows and knees free; both used to be affected	First at 15.	
9	M	20—25	Almost universal, including palms and bend of elbows	Several years.	
10	F	18	Elbows, knees, and limbs	Ditto	Mother and two sisters.
11	M	4	<i>Ps. guttata</i> ; elbows and knees	?	
12	M	28	<i>Ps. guttata</i> ; elbows and knees not affected	5 or 6 years; began at Gibraltar.	
13	F	6	Ordinary psoriasis; elbows and knees	?	
14	F	11	Ditto	4 years; every spring.	
15	M	21	Ordinary, but extensive and obstinate	1 year	Father and mother died of phthisis. ¹
16	F	18	Ordinary; scalp much affected	10 months	
17	F	38	Ordinary, extensive, including palm and soles, and a patch of <i>Ps. lingua</i>	6 months	
18	F	7	Ordinary; elbows and knees	?	
19	F	5	Ordinary, including scalp	2½ years	Father.
20	F	9	Ordinary; elbows and knees	?	
21	F	40	Knees, thighs, and calves; nowhere else	One attack 7 years ago in the same situation.	
22	F	44	Ordinary	4 years.	
23	F	8	Ordinary	Second attack	Father and brother.
24	F	24	Ordinary	?	Mother of 25.
25	M	8	<i>Ps. punctata</i> ; trunk only	?	Son of No. 24.
26	F	26	<i>Ps. guttata</i> ; limbs only	Frequently recurs.	
27	M	20	Ordinary; scalp	?	
28	M	15	Ordinary; legs only	?	
29	M	27	Ordinary; scalp	?	
30	F	14	Ordinary	7 years; spring and autumn.	
31	M	50	<i>Ps. guttata</i> ; trunk. Acute eczema of head and neck	?	
32	M	20—25	Severe and general, chiefly <i>Ps. gyrata</i> ; not specially on knees and elbows	First attack; several months. ¹	

¹ This patient was himself a robust well-built young man, a working engineer. He had been free from all affections of the skin until the spring of 1879. He was treated elsewhere from September to December with no benefit, first with iodide of potassium and then with small doses of arsenic. He completely recovered by February under full doses of arsenic and local application of tar.

No.	Sex.	Age.	Form and distribution.	Duration.	Family history, &c.
33	M	35	Ordinary, chiefly <i>Ps. guttata</i> ; trunk and limbs, including palms	Second attack; first at 30.	Mother.
34	M	18	Ordinary, including scalp	?	
35	F	39	Ordinary, very general; complicated with ordinary eczema, intertrigo of mammas and groins	Third attack; with each, lactation.	
36	M	28	Ordinary, complicated by a subsequent syphilitic eruption from a hard chancre	Several years; usually each spring.	
37	M	34	Ordinary, very general, not specially on elbows and knees	From childhood; worse in spring and autumn	
38	F	43	<i>Ps. gyrata</i> ; neck, throat, chest, and nape, down to scapulae	After each deli-very, i.e. twelve times.	Has had gout.
39	M	27	<i>Ps. nummulata</i> ; elbows, knees, abdomen	2 months; second attack; first last summer.	
40	F	21	Ordinary; elbows and knees	Third attack; 5 years	
41	M	20	Ordinary; extensive	Fifth attack since 9 years old.	
42	F	47	<i>Ps. guttata</i> ; both forearms	First attack; 6 months	
43	F	18	Ordinary; arms and legs	First attack; 2 months.	
44	M	40	Ordinary; limbs and scalp	4 years.	
45	F	44	Ordinary; arms and legs	Third attack.	
46	F	53	<i>Ps. guttata</i> ; knee and ankle; very irritable	First attack	
47	F	14	<i>Ps. guttata</i> ; arms, chest, back, and knees	Second attack; first when 11	
48	F	12	Knees and elbows	Repeated; every spring.	Father had a "disease of the skin."
49 ¹	M	20	General; guttata and diffuse	First.	
50	F	30	Elbows and knees	Second; first 18 months ago	
51	F	44	Arms, &c., palms, nails	Several years	
52	M	54	Ordinary; general	Since 14	
53	M	19	Ordinary	First at 17	Mother had the same.
54	M	10	Ordinary	First at 9	
55	M	58	Elbows, hips, legs	Several years	

¹ This patient had been previously treated for syphilis without benefit. The distribution was somewhat misleading, but he rapidly improved under arsenic.

² While this young man was under treatment, and the eruption gradually disappearing under solution of arsenic, of which he was taking *mvij* three times a day, there suddenly occurred an acute outbreak of numberless fresh spots of

The following is an anomalous case of disease, remarkable in more than one aspect, which I have with some hesitation regarded as an aberrant form of psoriasis.

CASE.—George T—, æt. 17, is a florid, fairly nourished lad, with the left leg wasted and contracted from obsolete disease of the hip. He never remembers being free from the affection of the skin for which he now seeks relief (Feb. 1, 1878). He believes it used to be on his face, and is sure that when he was a patient under Dr. Frederick Taylor, in 1876, his neck was affected. About Christmas time (1877) it spread over his abdomen and loins. The adjacent parts of the trunk and limbs have been the constant seat of the disease. It is now distributed over the shoulders, arms and forearms, flanks and abdomen, buttocks and thighs, with an imperfect symmetry. The scalp and face, ears, neck, legs, hands and feet, and genitals, are entirely free, and (excepting the head) appear to have been always so. The diseased surface is for the most part slightly injected, without pigmentation, papules, or other lesion, and with no evident cicatrices. Towards the margins, which are more or less gyrate, it becomes more red, somewhat raised, and covered with small, white, adherent scales, which in most parts form a series of concentric margins.

On removing these scales, a somewhat pigmented and injected surface is found beneath, without the least trace of moisture.

Though there is no pigmentation of the regions now affected there are maculæ on the chest and abdomen, where the patient states that the same eruption has existed. There is moderate irritation, no active symptoms. The colour is red or slightly purplish, without a coppery tinge. Careful investigation shows absence of all signs of either acquired or congenital syphilis. Repeated microscopic examination demonstrates that there is no fungus present. The sebaceous glands are apparently not affected.

After some trouble I found the mother, who is herself a healthy woman. She says that her husband is deaf, but free

Ps. guttata, chiefly on the arms, but also on the trunk and legs, where the original eruption was marked by little more than pigment spots. He was finally cured by taking him into the hospital, using tar thoroughly and pushing the dose of Fowler's solution to $\mathfrak{m}\mathfrak{xv}$ thrice daily.

from any disease of the skin. She has suffered from sore hands, apparently *eczema rimosum*. She has four other children, one older and three younger than my patient. One only of them has any affection of the skin. This girl afterwards also came under my care.

M. T—, æt. 15, a healthy, well-developed girl. Like her brother she was born healthy; but he was first attacked by his present complaint when about seven years old, and she at the same age or perhaps a year later. The skin of face is somewhat red and rough. The shoulders, upper arms, and chest are covered with a reddish, serpiginous, scaly eruption, with smooth, rather pale patches inside the circles, or the gyri which are made by their confluence. There is no moisture, no cicatrisation, and the disease is in all respects identical with that described in her brother. Here, again, there was no fungus present, and absolutely no evidence of syphilis, hereditary or acquired.

Putting aside the diagnosis of lupus erythematosus, from the absence of nodules, of sebaceous implication, and of cicatrisation; and that of tinea, from the absence of spores—it appeared to me that the only name to give it, if any known disease, was that of psoriasis. The appearance in brother and sister, the dryness and scaliness, the mode of progress, the inveteracy and proneness to exacerbations, as well as the itching and the pigmentation following its involution, all weighed with me in this decision. I accordingly prescribed Fowler's solution in gradually increasing doses, and George T— continued his medicine with great regularity up to June (1878), when he was taking fifteen or twenty drops daily. He then came to show how much better he was, and indeed the eruption had almost but not entirely disappeared. He then gave up attending, and I did not see him until the end of August, when he came again almost as bad as before. Again I lost sight of him till the 8th of October, when he resumed his former prescription, and again improved. After three weeks, thinking himself better, he gave up treatment once more. He came towards the end of November with increase of the disease and fresh circinate spots on the chest; there was also more irritation than before. I then ordered a larger dose of arsenic and Liquor Carbonis Detergens locally; but as soon as he improved

again he ceased to attend, and I have not been able to see him since, though I have heard of him at other hospitals.

So far, the result of treatment would seem to confirm the diagnosis. I should add, that before seeing me he had been treated with arsenic with apparent benefit, that he had taken *Liq. Hyd. Perchl.* for several weeks without any effect, and used sulphurous acid lotion with the same negative result.

On the 18th of November, 1878, I showed both this patient and his sister at the Hunterian Society. Several experienced dermatologists, who then saw the cases recognised their resemblance in certain points to erythematous lupus, syphilis, and tinea, but all agreed that it was none of these. Mr. Hutchinson, to whom I sent the patient, told me that the nearest resemblance to it he had seen was in a young man from Canada, the subject of an eruption which had existed from birth; it was not syphilis or tinea, but resembled ichthyosis and resisted all treatment.

I have no doubt that George T— is the same patient whose case was described to the Clinical Society two years later by Dr. T. C. Fox ('Lancet,' November 20th, 1880), as persistent gyrate erythema. Whether erythema should be extended to a scaly eruption which persists for years is a question of terms; but if erythema is used as a synonym of dermatitis, the question still remains as to the pathological nature of the disease.

I am indebted to my friend Dr. Cavafy for the suggestion that my case might, perhaps, be regarded as coming under what Dr. Duhring has described as *Pityriasis maculata et circinata*: see his treatise on 'Diseases of the Skin,' 2nd ed., 1881, p. 305.

IV. LICHEN AND OTHER PAPULAR FORMS OF DERMATITIS.

If we examine the descriptions given by Willan and his followers of the three classical papular diseases, lichen, prurigo, and strophulus, with their several species, we find very little which corresponds with the necessities of modern pathology.

Willan defines LICHEN as "an extensive eruption of papulæ affecting adults, connected with internal disorder, usually terminating in scurf, recurrent, not contagious." Except the first anatomical character and the last negative one, there is nothing here to help us. STROPHULUS appears to mean nothing but papules occurring in children, which are not contagious, i.e. neither scabies nor measles. The term is deservedly neglected at present; and the varieties of "red gum" are referred to papular erythema. Bateman's seven species of lichen are *L. simplex*, *pilaris*, *circumscriptus*, *agrius*, *lividus*, *tropicus* and *urticatus*. The last would seem to be indistinguishable from *Prurigo infantilis* as described by Hutchinson and other writers, which is generally accompanied with more or less consecutive erythema and urticaria, the wheals and the diffused redness being alike the result of scratching, and not the origin of the papules.

The term lichen with a former generation included what we now call papular syphilis. It was also applied to the acuminate papules most marked on the extensor surface of the upper arm, the calf, the outside of the thigh and the buttocks in brawny men (*Lichen pilaris*, *pityriasis pilaris* of Devergie); these are due to accumulation of dry sebum and dead epidermis in the large sebaceous ducts and hair sacs of these regions; and they sometimes form minute centres of inflammation and even of suppuration. The condition is removable by friction with soap and hot water, and scarcely deserves a pathological recognition.

The same condition, however, occasionally occurs in a more remarkable form as the following case shows:

Lichen pilaris of limbs in a child.—A thin, delicate looking girl, twelve years of age, was brought to me for "roughness of the skin." There was slight branny desquamation without redness or seborrhœa of the face and scalp; but the limbs were covered with small, hard, pale, pointed papules, more readily felt than seen. Each corresponded to a hair-sac, and resembled the ordinary lichen pilaris of the thighs and legs in adult males. The papules, however, were harder and closer set, and affected not only the extensor aspect of the limbs, but the soft skin of the elbow and ham, covering in fact the whole of the thighs, legs and arms. The back and trunk generally were free.

The affection was not congenital and had only appeared since the child was eight years old. Though thin and pale, the patient had no disease of the lungs, lymph glands, joints, &c., which could enable one to call this *Lichen scrofulosorum*, and it will be seen that the eruption did not correspond with Hebra's description of that affection. On the other hand, it was obviously different from *Lichen ruber*, or any inflammatory disease, and would pathologically seem rather to be allied to pityriasis, xeroderma, and the rough dry condition of the skin in children which is connected by intermediate grades with ichthyosis.

Lichen agrius, with small vesicles and liability to terminate in a chronic pustular disease, is certainly a form of acute eczema. So is *L. tropicus*, of which I have given a case above. *L. lividus* is purpura affecting the vascular hair sacs. There remain only *L. simplex* and *L. circumscriptus*. The acute course and slight desquamation of the former seems to mark it as a true erythema. The latter alone of all the species would probably be admitted as lichen by a modern physician.

The acute papular eruptions, especially on the limbs, which have been described as lichen by later writers, may be fairly classed under papular erythema, when, as is often the case, they justify the name by their rapid course, their frequent recurrence, their appearance in patches, their distribution, and their connection with gastric disturbance. Lastly, I would recognise as eczema all papular eruptions which are chronic in course, diffused and spreading in their distribution, and localised in the flexures of joints, the back of the ears, and other favourite eczematous sites; which are attended with itching rather than pain, which lead to infiltration of the skin, and which either follow or precede ordinary moist eczema.

The species of *PRURIGO* described by Bateman are: 1. *Pr. mitis*, mostly affecting young persons, and sometimes ending in "contagious scabies." 2. *Pr. formicans*, occurring in adults and affecting the whole of the trunk and limbs, except the feet and palms, "but most copious in those parts over which the dress is tightest." 3. *Pr. senilis*, in the course of which "pediculi are not infrequently generated." 4. Local prurigo,

differing from the above varieties in not being papular, and only resembling them in itching, viz. *Pr. præputii*, *Pr. pubis*, *Pr. urethralis*, *Pr. podicis*, and *Pr. pudendi*.

The first of these is the result of irritation from dirt, and the second of pediculi. The third is not prurigo at all, for it is secondary to affections of the bladder, and modern pathology separates those affections in which papules if present are the effect and not the cause of itching, the direct traumatic result of scratching, and names them "*Pruritus*." *Prurigo podicis* and *Pr. pudendi*, still so called by some French writers, is named *Lichen podicis* by Hardy and *Eczema ani* by Bazin. It is the well-known chronic, intensely irritable dermatitis, usually papular, but often made eczematous by scratching, which affects the vulva, the perinæum, or the anus in persons past middle age, and has been included above under Eczema.

Much of what was called *Pr. formicans* and nearly all *Pr. semilis* was probably due to pediculi corporis and now known to be curable by destruction of these vermin.

There remain two valid diseases called Prurigo. One is Hebra's prurigo, which I have seen at Vienna, and which appears to occur occasionally in its full severity in America. Such cases, however, seem to be only remarkably severe, and possibly over-described, cases of what we see in England, obstinate and chronic prurigo of adults, with thickened and pigmented skin, but without the characteristic localisation of *Pr. pedicularis*, and unaffected by parasiticides. Such cases are in fact recognised by Hebra as *Prurigo simplex*. The other distinct form of disease is that called *Prurigo infantilis*, *Strophulus*, and *Lichen urticatus*, an obstinate eruption of large, flat, rather pale papules, chiefly confined to the trunk and adjacent part of the neck and limbs, and always avoiding the face, scalp, hands and feet, attended by intolerable itching, subject to periodical exacerbations (whence it has received such names as summer prurigo), and rarely seen before weaning or after the approach of puberty. It is possible that some of the worst of these cases may go on to the prurigo simplex of adults, or even to its severer forms; but as observed in children the skin is not thickened, pigment is not increased, and the hands and feet are markedly exempt. That some of these cases are due to irritants, and especially to fleas and other vermin, is possible,

but there must be more than the direct result of such irritations to produce prurigo; for they often seem to have little effect, and when this follows it is usually an erythema, which disappears when the cause is removed. In contrast with *Prurigo pedicularis* we may therefore fairly call this disease idiopathic. I have seen one marked case of this form of prurigo following weeks after an attack of varicella, as described by Mr. Hutchinson.

The following are brief notes of cases of prurigo as above defined:

CASE 1.—M., æt. 43. January. Papules on shoulders, forearm, and loins; slightly on abdomen. Head and face and limbs free. Large, flat, discrete papules, with scratch marks and slight erythema. Severe itching. Subject to it since childhood. Two months later the forearm, buttocks, and thighs were also affected. Treated with quinine without apparent benefit. No pigmentation. Not due to any discoverable irritant.

CASE 2.—M., æt. 42. July. A similar case, but more extensive, the limbs as well as the trunk being affected. Back and shoulders less so than loins, buttocks, and limbs. Head, face, hands, feet, and genitals alone free. Papules separate, and many capped with dried blood. Scratch marks, with a good deal of erythema and urticaria. Seven years' duration, always worse in summer and better in cold weather.

CASE 3.—F., æt. 15. October. Generally distributed; papular. Was, during the summer, a patient of Dr. Fagge, who treated her with marked success by full doses of quinine. Three grains of the sulphate taken three times a day appeared again to be extremely useful, and after a few weeks' treatment she was again freed from her troublesome complaint.

CASE 4.—M., æt. 12. May. Large scattered papules, some with bloody tips, over back, nates, thighs, arms and forearms. Six months, from November to May. A thin pale boy.

CASE 5.—M., æt. 12. Papules and scratch marks, without pigmentation, on back, loins, thighs, and (slightly) on upper arms. A pale thin boy. *Pediculi corporis*.

CASE 6.—M., æt. 12. Scattered pruriginous papules. Great benefit while taking quinine and using hydrocyanic acid (ʒiv of the dilute acid to a pint) as a lotion,

CASE 7.—M., æt. 6. May. Small, colourless, almost invisible papules, none closely set, with raised patches of urticaria. Loins, abdomen and limbs. Head and face, shoulders, and hands and feet quite free. Has lasted two years. Impetigo capitis before.

CASE 8.—M., æt. 1½. Large, flat, pale, discrete papules over back and abdomen. Some urticaria. Slight eczema of one axilla. A fair, well-nourished child. No source of irritation discovered.

CASE 9.—M., æt. 4. January. Papules and scratch marks over back and shoulders. Began last summer.

CASE 10.—M., æt. 3. Small discrete papules, with a few vesicles and scratch marks, but no erythema. Abdomen and trunk generally; arms and legs also affected. Excessive irritation. A healthy child. No irritant discovered. Treated with quinine without benefit. After several months, gradual improvement independent of treatment. Next August relapse.

CASE 11.—M., æt. 2. March. Had chicken-pox about a year ago. For three months has suffered from exceedingly irritable papules, with pustules. The disease was first called *Lichen urticatus*, then "Erythema pustulosum et bullosum." When I first saw it, I suspected scabies, but a careful search failed to discover not only the acarus (a common failure in the case of infants), but any runs, vesicles, or other characteristic lesion. Moreover, the distribution was unlike that of scabies, the papules being irregularly scattered over the trunk and limbs, and there was no other case. The pustules and blebs were due to the child's scratching, and were accompanied with wheals of urticaria. It continued very obstinate for several weeks.

CASE 12.—M., æt. 3. January. Colourless papules, with slight erythema on chest, hips, &c. Head and limbs free. A well-nourished, healthy child. No trace of vermin or other irritant.

CASE 13.—M., æt. 2. Papules with slight erythema, without wheals, over loins, back, arms, thighs and legs. Five months' duration; began in September.

CASE 14.—F., æt. 4. Papular rash on abdomen, flanks and loins; head and limbs free. Summer prurigo. Well during the winter, and came again the next spring.

CASE 15.—In a child, set. 2. June. Pale scattered papules, one only having become vesicular, over the abdomen, arms, thighs and legs. Has existed since birth. No eczema, impetigo, erythema, or urticaria. Very irritable.

CASE 16.—M., set. 7 months. August. Large pale discrete papules, chiefly upon the trunk. Four months duration. Very irritable.

CASE 17.—M., set. 15 months. May. Rather small papules, scattered over abdomen, back, and arms. Much urticaria, which was more prominent than the prurigo; and papular eczema of one arm. Appeared a fortnight before with the warm weather. A fat, healthy child. No appearance of flea bites or other irritants.

There is one form of papular dermatitis of which we find no account in the earlier works on dermatology—I mean *Lichen planus*.

No one who has seen a well-marked example of this affection can doubt the accuracy of Mr. Wilson's original description of it. The raised, flat patches—miniature plateaux rather than plains—their dull, glistening surface, deep purple-red colour, and the frequent marks it leaves behind, are very characteristic. The localisation is not constant. Most frequently, perhaps, the back of the hand and wrist are the seat of the disease; scarcely less so the leg or thigh, and the patches are not confined to the extensor surface of the limbs. The trunk is also not unfrequently affected. It does not seem to have been observed in children. It is usually said to be more frequent in women than men, and in thirty cases collected from various sources I found eleven were men and nineteen women; but the numbers are too small to be conclusive, and the difference too slight to be important. It is, however, worth noting that almost all Hebra's cases occurred in men.

As in psoriasis, the amount of itching varies greatly; some patients feel scarcely any irritation, others complain greatly of this symptom, and scratch marks or secondary dermatitis sometimes confirm their complaints. The course of the affection is always chronic. Arsenic and local application of tarry compounds are sometimes very quickly efficacious, but I have sometimes found the cure tedious.

I have most often seen *Lichen planus* mistaken for syphiloderma.¹

There can be no doubt of the close alliance (illustrated by one of the cases given below) of this affection to certain other forms of papular dermatitis, especially to what Hebra describes as *Lichen ruber*. They are both essentially papular, both chronic, both dark, both irritable; they are somewhat similar in distribution, and they sometimes occur together. Indeed, Mr. Wilson recognises the close resemblance or identity of his *Lichen planus* with the disease previously described by his colleague in Vienna, and frankly yielded him the priority. Nor can we fairly question the relation of *Lichen planus* to psoriasis, which has been so well supported by Mr. Hutchinson. It is very seldom that the resemblance is one of appearance or distribution; but it depends on the clinical features of dryness, chronicity and irritability, the common character of solid hard papules becoming afterwards scaly, the liability to pigmentation, the readiness to return, and the reaction to the same therapeutic measures. All these physiological characters point to a true kinship. A case exhibited at the Pathological Society by Mr. Marrant Baker, in the session 1880-1, showed the occasional difficulty of diagnosis between psoriasis and *Lichen planus*.

On the other hand, I would, at present, separate *Lichen planus*, and even *Lichen ruber*, somewhat sharply from other so-called species of "lichen." The word by itself has come to mean little more than a chronic eruption of papules, and I doubt whether we can at present use it to any better purpose.

The following cases of *Lichen planus* seem worthy of being put on record.

CASE 1.—A man, aged 80, presented himself among the out-patients with eight or nine flat, raised, slightly-scaly red patches, from a pea to a threepenny-piece in size, situated on the back of the wrists, the forearm, and the dorsum of the hand. On the right leg there were several similar *plaques*, which had united, and here there was a good deal of ordinary dermatitis,

¹ So Dr. Duckworth ('St. Barth. Hosp. Rep.,' vol. viii), who also agrees with me in regarding *Lichen planus* and *Lichen ruber* rather as closely allied than identical.

set up by scratching. The eruption had lasted three months, and had never appeared before. Next week he came with a fresh crop of scattered papules upon the inside of the left thigh. He was ordered Fowler's solution, but disappeared before he was cured.

CASE 2.—A lady, aged about 45, came to me, having been previously treated with mercury for what was supposed to be a syphilitic eruption. For three months she has noticed small red patches on her hands and feet, and they have gradually increased. They began as "pimples," and a few separate papules are still present, but the lesion consists chiefly of raised, flat, bright red elevations of the skin, covered with faint indications of minute scales. They do not spread, but fresh papules appear and coalesce. The parts affected are both hands, on the backs, between the fingers, and on the palms; less so the feet, including the soles. The eruption is very irritable. There are two smooth patches inside the cheek and on the dorsum of the tongue (*psoriasis linguæ*). No other lesion; healthy aspect. History of a rash several years ago which affected the arms, legs, and waist. The colour and the uniformity of the eruption, the irritation, which was decided though not severe, the account of a previous eruption which was pretty certainly not venereal, and the absence of all other signs of syphilis, convinced me that the affection was not of that character, notwithstanding the suspicious localisation and the curious coincidence with an affection of the tongue, which is often mistaken for a specific lesion. I accordingly prescribed five drops of Fowler's solution three times a day and only cold cream locally. A week later there was slight irritation of the conjunctiva, and the rash was much improved, paler, and no longer irritable; while no fresh papules had appeared. But after another week fresh spots with fresh irritation were observed on the forearm and at the bend of the elbow. Somewhat later the same papular eruption affected the legs and abdomen, and was accompanied by great itching. The patient, however, persevered in the use of arsenic, to which I had added *Liquor Carbonis Detergens* as an ointment (3ij to the ounce of vaseline), and wrote to me from the country, in October, that she had lately improved. The final result I have not been able to ascertain.

CASE 3.—A very similar case to this last one is the following :—A stout, healthy-looking woman of fifty-three came to the hospital with a chronic lichenous eruption on the back of the hands, the forearms, upper arms, ham, and thighs. Besides papules, there were the flat, smooth-topped, scaly, raised patches of Lichen planus, and here and there the scales were so much developed, and the affected surface so large, that the case looked like psoriasis. There were flat, smooth patches on the tongue and cheeks (*psoriasis linguæ*), but no sign of syphilis. Similar treatment, by arsenic internally and tar ointment locally, succeeded much better than in the former case. Maculæ remained after the cure was complete.

CASE 4.—A case, of which I have only a short note, occurred in a woman of thirty-eight, who came among my out-patients with "Lichen ruber, papulatus et planus" affecting both forearms and wrists on the flexor side. Colour dark red, smooth surface, irritable. The affection had lasted three months, and had never occurred before. I treated it in the same way as the last, but did not learn the effect.

CASE 5.—A remarkably strong, large-framed labourer, 35 years old, came with four well-marked patches of Lichen planus on the back below the angle of the left scapula and somewhat lower down on the right flank, and on the flexor surface of the right forearm. There were papules, separate as well as coalesced into the raised *plaques*. The colour was a dark purplish red, the surface glistening and covered with small, fine scales. In a few weeks, under tar ointment locally and arsenical solution, increased to eight drops three times a day, internally, the eruption disappeared, leaving decided pigmentary stains behind.

CASE 6.—A patient of mine, suffering from mitral insufficiency as the result of rheumatism, an otherwise healthy young man, somewhat under thirty, showed me an eruption which he suspected to be syphilitic, although he had never had a chancre. It had lasted several months. There were scattered papules on both legs, some of them covered with small scales, and patches looking like *psoriasis nummulata*. The colour was a deep red. There was great itching. The knee-caps were free, the parts affected being the shin, calf, ham, and adjacent part of thigh. The arms were also free. I ordered Liquor arsenicalis, five

minims after each meal. There was some old, dry, chronic dermatitis of the hands, the remains of what in former years seemed to have been ordinary eczema. A few weeks later the patches were more distinctly raised, flat, shining, and fissured, and the scales more scanty and minute. As Fowler's solution produced disturbance of the stomach, I now ordered five drops of *Liquor Sodæ Arseniatis* three times a day, with perseverance in the use of the ointment. When the smell of this last became insupportable to the patient, I substituted a strong ointment of *Liquor Carbonis Detergens* (ʒiv to ʒj of vaseline), and increased the dose of Pearson's solution to seven minims three times a day. The eruption had been steadily fading, no fresh spots appeared, and the cure was complete in about eight weeks; slight pigment stains were left behind.

CASE 7.—A man, aged 21, but looking older, of dark complexion and strongly built, came as an out-patient, with characteristic *Lichen ruber* and *Lichen planus* affecting the face, chest, back, and trunk generally, as papules; and the elbows, forearms, and thighs, as raised, flat patches. The colour was dark, but not coppery; itching not severe. He recovered under treatment with tar ointment and moderate doses of arsenic.¹

V. DERMATITIS EXFOLIATIVA. PITYRIASIS RUBRA.

The species of *Pityriasis* as defined by Bateman are none of them entitled to permanence. *P. capitis* or dandruff is in most cases *seborrhœa sicca*, in others a slight local dermatitis; an *eczema squamosum*, often (as he remarks) due to want of cleanliness, and removable by soap and water, but apt, if neglected,

¹ I may refer students of this remarkable form of disease to the following descriptions and cases:—Wilson, 'Diseases of the Skin,' 6th ed., 1867, p. 156; Hillier, Tilbury Fox, 'Brit. Med. Journ.,' April, 1871, and in his 'Text-book,' p. 144; Hilton Fagge, in vol. xv, of the present series of these Reports, p. 341; Liveing, 'Hand-book,' 2nd ed., p. 138; Hutchinson, 'Lectures on Clinical Surgery,' p. 207. Dr. R. W. Taylor, of New York, has published four carefully observed cases of the disease in the 1st vol. of the 'Archives of Dermatology,' which show that its features in America resemble the English disease. We have two models of it in our Museum, Nos. 259, 260.

"to degenerate into Porrigo," i. e. to become pustular. Occasionally it is Psoriasis of the scalp, the scales being small and mixed with sebum owing to the locality. *P. versicolor* is a parasitic disease. *P. nigra*, observed by Willan in children born in India, was not identified by Bateman, nor I believe since. A case of Alibert's, which Devergie calls Pityriasis nigra with prurigo, was apparently Prurigo pedicularis with pigmentation and leucodermia. The fourth and last species, *P. rubra*, "resembling Psoriasis diffusa," denotes like it a stage in the involution of eczema. *P. rubra* of Cazenave seems to be only *P. versicolor* with more irritation than usual.

The word Pityriasis denotes, as its etiology implies, a branny, furfuraceous desquamation; and if we continue to use the term it is only as "roseola," "erythema," or "herpes" to denote a certain anatomical condition, without deciding upon its cause or predicting the event.

But the specific term, *PITYRIASIS RUBRA*, is now used no longer to denote such desquamation occurring on a red skin, as in eczema or scarlatina, but to signify a substantive disease. This application was made by Devergie in 1854. In his 'Traité pratique des Maladies de la Peau,' p. 263, we read:—"PITYRIASIS RUBRA.—Je place auprès de l'eczéma l'histoire de cette maladie, à cause des difficultés de diagnostic qu'elle présente, et de sa grande analogie de forme avec cette affection."

He describes the disease as beginning with an erythematous redness, usually on the chest or flexor surface of the limbs, and spreading rapidly, with a well-defined margin, deep colour, abundant scales, and more or less thin serous discharge. It covers the whole body,¹ is very obstinate, lasting for months, and occasionally proves fatal by exhaustion and diarrhoea. As a rule, however, patients slowly recover. Relapses are frequent. Devergie admits the difficulty of distinguishing this new disease from eczema, and bases the diagnosis on the following points, which I will put in a tabular form.

¹ "C'est la seule affection qui, avec le psoriasis aigu, puisse envahir à la fois toute la surface de la peau de l'homme" (loc. cit., p. 264).

Eczema.

Bright red colour.
 Border ill defined.
 Is never universal.
 The skin is not thickened.

Itching severe.
 Secretion stiffens linen.

Scales small, adherent, and
 only form during involution.

État ponctué.

Pityriasis rubra.

Rougeur foncée.
 Sharply-marked border.
 May affect the whole skin.¹
 The skin, and even the sub-
 cutaneous fascia are thickened.
 Less itching, more burning.
 Secretion thin, and does not
 stiffen linen.

Scales abundant, readily de-
 tached, and present from the
 first.

No red secreting points
 under the scales.

Devergie adds :—" Cette maladie ne se montre guère que vers l'âge de quarante à quarante-cinq ans. . . . on l'observe plus souvent chez la femme que chez l'homme." He ends his account of the disease by giving two cases in which "*pityriasis rubra* se transforma en pemphigus." The description of these cases resembles that of the Pemphigus foliaceus of Cazenave (which is not mentioned by Devergie, though it had been shortly before described and figured), but it does not appear that any bullæ formed, only a thick "mucous" liquid of faint foetid odour was secreted under the scales. One of these patients was a woman of sixty-one, who recovered; the other a man of fifty-two, who was still under treatment when the report was made, eight months after his admission to St. Louis, and five years after the beginning of the disease.

Dr. McGhie narrated, under the title "*Pityriasis rubra acuta*,"¹ a rare form of skin disease," a case which he rightly regarded as coming under the description given by Devergie ('*Glasgow Medical Journal*' for January, 1858, p. 421). This was, I believe the first published in this country, and intervened between Devergie's work and those by Hebra and by Wilson. The case was in a young man: it began with ordinary vesicular eczema of the elbow, rapidly spread as a dry red desquamating dermatitis to the whole body, and ended in recovery in rather more

¹ It must be observed that *signa*, to which Hebra afterwards took exception, refers to the onset not to the duration of the disease. All Devergie's cases are chronic.

than three months. The desquamation was branny (pityriasis in the literal sense), and there was some pyrexia. Dr. McGhie carefully distinguishes his case from eczema and from psoriasis.

This same patient was brought forward by Professor Gairdner, of Glasgow, seventeen years later ('British Medical Journal,' March 13th, 1875). He had suffered repeatedly from returns of the same disease, each lasting for some months and affecting the nails as well as the skin. In the last attack, from October, 1874, to February, 1875, the temperature was found to vary from a little under 100° up to 103·8°.

In the first volume of his 'Hautkrankheiten' (1860), Hebra adopts the term of Pityriasis rubra in Devergie's sense. His description, based upon three cases, agrees essentially with that just given, and he especially lays stress on the universality of the disease, its remarkable red colour, its great obstinacy, the dryness and desquamation, and the absence of itching. On three points, however, Hebra's account differs from that of Devergie. He makes infiltration of the skin the characteristic sign of chronic eczema rubrum, and its *absence* the peculiarity of pityriasis rubra. Instead of an abundant formation of large, thin, easily detached scales, Hebra speaks of *ganz unbedeutende Schuppenmengen*; and again of *geringe Schuppenbildung, ganz unbedeutende Abschuppung*. He omits all mention of moisture. Moreover, in his experience the prognosis was more unfavourable than in Devergie's; for the three cases he had observed all died. In his table of diagnostic points he contrasts the moisture of eczema with the dry scales of P. rubra, the papules of eczema with their absence in P. rubra, and the irritability of eczema with the constitutional symptoms of P. rubra.

The discrepancies in the accounts of these two eminent observers show the difficulty of making positive and conclusive statements from a small number of cases, for there can be no doubt that Devergie's and Hebra's cases were both distinct from eczema and both examples of the same disease. Further experience has shown (in Vienna as well as elsewhere) that the Pityriasis rubra is far from constantly fatal. Infiltration of the skin is not an invariable character of either eczema or Pityriasis rubra, but I have certainly found it present in almost every case of chronic eczema, and much less marked, or absent, in those of Pityriasis rubra which I have seen.

How Hebra can have made so little of the abundant desquamation can only, I think, be explained by the effects of his treatment: lukewarm baths continued for hours and the use of softening ointments, "verursachten insoferne eine Veraenderung, weil dadurch die Epidermismassen (so the Schuppenbildung must have been considerable) transparenter und die Haut geschmiediger wurde."

In 1861 Dr. Wilks described, in the seventh volume of the present series of these 'Reports,' a case of "General Dermatitis," acute, universal, red, dry, and scaly, the desquamation being very abundant and including the finger- and toenails. It lasted two months, and ended in complete recovery.

In 1867 Mr. Erasmus Wilson described three cases, which he identified with the *Pityriasis rubra* of Hebra, and proposed the names *Eczema foliaceum*, *Pityriasis foliacea*, or *Pityriasis foliacea rubra*.

The second case, termed *Psoriasis squamosa rubra* (*psoriasis* in Mr. Wilson's language meaning at that time dry scaly *eczema*), occurred in an old lady of 71; it was confined to the hands, and might be fairly regarded as obstinate *eczema manuum*.¹ But the other two cases, both in old men, are typical cases of *Pityriasis rubra*, in their universality, redness, dryness, and profuse desquamation, as well as in the uniformity of the lesion, and the absence of severe irritation and constitutional disturbance. One of these patients died of an attack of bronchitis, the other lived to recover of his disease.

Hebra did not admit that Mr. Wilson's cases were *Pityriasis rubra* in his sense of the word. The chief points of difference are in the scanty desquamation and the ill end of the three cases observed in Vienna. Dr. Hans von Hebra has, however, since published three cases (1876), which were presumably

¹ The obstinacy of the disease is attested and possibly explained by the following list of remedies given without success during ten months—An arsenical course of three months; a course of Donovan's solution for three weeks; bichloride of mercury for one month; nitro-muriatic acid with a bitter; small doses of sulphate of magnesia with quinine, with nitrate of potash, and with colchicum; iodide of potassium with colchicum; citrate of iron and quinine; liquor cinchona with ammonia and with sulphuric acid; gentian with soda; and various remedies besides. Meantime were applied locally oxide and chloride of zinc, acetate of lead, ammonio-chloride, nitrate, and nitric oxide of mercury, sulphur, iodide, carbolic acid, and tar.

recognised by his father as genuine pityriasis rubra. Two were in men; in one desquamation was profuse and in large flakes, in the other it was less abundant; both patients died of advanced phthisis, with tuberculosis of several organs. The third case began with impetigo of the scalp in a woman of 64, and agreed in the characters of universality, redness, dryness, and desquamation. She left the hospital after two months' unsuccessful treatment. Other cases have since been published by the late Dr. Hillier, in 1864,¹ as *Pityriasis rubra*, and by Dr. Fagge in these 'Reports for 1876,'² as "*Eczema squam-
osum universale seu Pityriasis rubra.*"

Mr. Wilson in his "*Lectures on Eczema*" (1870), describes the disease at length and gives an additional case. It occurred in a young man of 28, and was developed out of an ordinary chronic eczema. It showed the characteristic features of universality, deep red colour, dryness, absence of itching, and profuse exfoliation of large, thin scales, which Mr. Wilson compares to dried hops. There was decided infiltration of the skin, and the nails were affected. The disease lasted from October to January after an acute onset; by the end of three months the patient was entirely free from it.

In these lectures, Mr. Wilson proposed to substitute the title "*exfoliative dermatitis*," or *ECZEMA EXFOLIATIVUM* for *pityriasis rubra* or *pityriasis foliacea*. *Pityriasis* was an ill-chosen word, for the desquamation is anything but branny, but the term is now established and is distinctive. Moreover, most pathologists deny that the disease is eczema, and I think on good grounds.

The late Dr. Tilbury Fox (writing in 1878),³ while agreeing with the descriptions of Devergie, Hebra, and Wilson, not only separates *Pityriasis rubra* altogether from association with eczema, but maintains that it is not truly a dermatitis at all. Dr. Living,⁴ on the other hand, agrees with Dr. Fagge in regarding it as only a peculiar form of eczema. He admits the absence of visible exudation, but has found traces of it on the under surface of the large thin scales. He also describes the cutis as not thickened by inflammatory infiltration. In two

¹ 'Handbook of Skin Diseases,' p. 101.

² 'Guy's Hospital Reports,' 3rd series, vol. xiii.

³ 'Skin Diseases,' p. 252.

⁴ 'Handbook of the Diagnosis of Skin Diseases,' p. 99 (1878).

cases he had observed albuminuria. One of the patients recovered, the other died of chronic Bright's disease. In a third case, under Dr. Henry Thompson, there was no albuminuria; but the patient died, and *post mortem*, no organic disease was discovered.

Mr. Hutchinson, in the following year (1879), published three most interesting lectures on Pityriasis rubra.¹ He admits that the absence of liquid exudation and of thickening of the skin distinguish it anatomically from eczema, but regards its essential features as universality and resistance to treatment. He would therefore regard it as a type of a group of affections differing in their anatomical features and including "Pemphigus foliaceus, certain rare cases of diffuse eczema and psoriasis, which end fatally, some forms of senile psoriasis palmaris, some of onychitis, and some of lichen psoriasis" (*i.e.* Lichen planus and rubra). As to the pathology of this group, Mr. Hutchinson compares them with generalised destructive inflammation of the joints, and thinks that like that condition they will prove to depend on a neurosis. He argues that their symmetry and universality are conclusive against Pityriasis rubra and its allies having a local origin. "We have therefore to choose between the blood and the nervous system, and in the entire absence of any proof of implication of the former I prefer to suspect the spinal cord." To my mind, I confess Pityriasis rubra is symmetrical because it is universal, and is universal because the skin is universal. The universality is no doubt an important feature of the disease, but it belongs also to Ichthyosis, which is totally different in its natural history no less than its anatomy. Moreover, eczema may be more nearly universal than many undoubted cases of Pityriasis rubra without losing its characters of eczema. The resistance to treatment is another important feature, but this also applies to several other diseases of the skin which have no other bond of union. And reference to the table at the end of this paper will show that most of the cases recorded have recovered, and that many have in the judgment of the recorders been cured by treatment.

As to the pathology of Pityriasis rubra, in the absence (as it seems to me) of any proof of implication of the blood or the

¹ 'Lectures on Clinical Surgery,' pp. 240—274.

nerves, I prefer to suspect the skin. For whatever else the disease may be, it is certainly a dermatitis; and there seems to be no reason why the living cells of the skin should not be liable to idiopathic inflammation as much as those of the mucous membranes, the kidneys, or the lungs. Beside the clinical evidence we have also histological facts proving that *Pityriasis rubra* is a true dermatitis, which have been observed independently in Vienna and in London.

A microscopic investigation of the skin made by Dr. Hans von Hebra showed that in a fatal case of the disease, which had lasted a year, the whole of the cutis, papillæ, and deep layer, with part of the subcutaneous fascia, was filled with leucocytes. In the other fatal case, which had lasted several years, the condition was very different; it resembled cicatricial tissue. The Malpighian layer of epidermis was thin, and its cells shrunken; the papillæ were also atrophied, and only few remained; the papillary layer was represented by a thin layer of connective tissue, under which a thick layer of yellowish-brown elastic fibres with abundant granular pigment represented the deep layer of the cutis. No sweat glands could be found, and but few sebaceous glands.¹

In the same year (1879) in which Mr. Hutchinson's lectures were published Dr. Buchanan Baxter published a valuable paper on the subject in the 'British Medical Journal' for July 19th, under the title "General Exfoliative Dermatitis." He details five cases.

The first was a universal dull, red, dry eruption, with infiltrated skin and profuse desquamation, and occurred in a little girl, six years old—a truly acute case, running its course in less than two months, with albuminuria and moderate pyrexia, and proving fatal by œdema of the lungs and diarrhœa. Beyond chronic peritoneal adhesions and bronchial flux, with emphysema and œdema, no lesion was found after death. The kidneys are not mentioned, and therefore we may assume they were not the subject of Bright's disease. Sections made of the skin showed slight swelling of the papillæ and enormous thickening of the cuticle, while the Malpighian layer, instead of being sharply-defined from the latter, passed very gradually into it, the intermediate "granular layer" of epidermis having disappeared.

¹ I quote this account from a report in Behrend's 'Hautkrankheiten,' 1879.

The second of Dr. Baxter's cases occurred in an infant six months old who, after suffering several weeks from ordinary eczema of the head and face, was attacked with universal dermatitis of a dull red colour, dry, and producing abundant large thin scales. It proved fatal in eight weeks.

The third case was in a woman, aged twenty-eight. It presented the characteristic features of Pityriasis rubra, and ended favourably after a course of between two and three months.

The fourth was a remarkable one. It occurred in a boy of seven, and was at first regarded as Lichen ruber; as it gradually spread over the whole body it assumed rather the characters of an "acute psoriasis," but on the whole Dr. Baxter regards it as belonging to the series of Pityriasis rubra or general exfoliative dermatitis. Notwithstanding its wide diffusion, dryness, and profuse desquamation, the fact that some parts of the body appear to have escaped, and the presence of papules, appear to me to be important points of difference. After three months' treatment (begun three weeks after the appearance of the disease) by warm baths, with arsenic and cod-liver oil internally, the eruption had disappeared, but the boy was weak and thin, and died a few weeks afterwards from some acute febrile affection. There had been no albuminuria during his illness.

Dr. Baxter's last case was one of pemphigus (apparently not syphilitic) coming on ten days after birth, which passed into general dry exfoliative dermatitis. The infant recovered in two or three weeks under arsenic. This, Dr. Baxter says, would probably be called a case of Pemphigus foliaceus; but he argues for the recognition of general exfoliative dermatitis as a common meeting point of the four "dartrous" or herpetic disorders which are curable by arsenic, viz. Eczema, Psoriasis, Lichen, and Pemphigus, whenever they become universal.

The objections to this ingenious hypothesis seem to me to be the following:

1. Eczema in its ordinary moist form may be universal or, at least, as nearly so as many of the cases which Dr. Baxter would include, without changing its character, and may so continue for years without either the peculiar anatomical structure or the physiological effects which characterise the typical cases of "general exfoliative dermatitis: witness my case in a lad who had suffered since birth from eczema (p. 215).

3. Though few, if any of us, in England have seen a case of universal and chronic Lichen ruber as Hebra described it, yet he carefully, and apparently with justice, distinguished it from *Pityriasis rubra*.

8. We may find the characteristic anatomical characters of exfoliative dermatitis or "*Eosema foliaceum*," not as a universal but as a local affection. Of this I shall give several examples presently (p. 272).

4. *Pemphigus foliaceus* is not ever, I believe, universal; it is almost limited to women; and its obstinacy and the considerable and often foetid discharge explain its constitutional effects: whereas some of the most marked cases of *Pityriasis rubra*, free from irritation, and almost from apparent inflammation of the skin, are yet productive of grave internal effects.

5. That the whole of the skin may be occupied by a thick scaly disease without interference with health is proved by many cases of Ichthyosis. I have now under my care, in Miriam Ward, a child whose entire skin, from head to foot, including the palms and soles and scalp, is occupied by the severest form of Ichthyosis cornea; and she suffers greatly from itching. Yet she has an excellent appetite, sleeps well, and is fat and firm in flesh. The urine is free from albumen and, in fact, she is not "bodily ill" at all.

The course of opinion upon the nature and limits of *Pityriasis rubra* has been even more divergent in France than in England. Some dermatologists accept Devergie's term in nearly his meaning. Basin writes of a "*Pityriasis rubra aigue généralisée qui s'étend à la presque totalité du corps*," and the abundant exfoliation of large scales which he describes confirms the belief that this is Devergie's, Hebra's and Wilson's disease; but he also describes a "*Herpétide exfoliatrice*," and the case so named by M. Guibout (No. 19 in the table at the end of this paper) is certainly one of *Pityriasis rubra*. Hardy describes *Pityriasis rubra* in Willan's, or rather, perhaps, in Casenave's, sense of the term, as a branny desquamation on a red skin, commonly occupying the head and neck, but "*quelquefois toute la surface du corps*." General symptoms not unfrequently accompany it, especially fever and digestive derangement. These last two characters recall Devergie's

¹ 'Leçons,' p. 125.

beginning to heal by desquamation. There is now no moisture anywhere, and the patient assures me that there never has been, even at the flexures of the joints. There is abundant exfoliation of small, white, branny scales, but none of the large, thin, hop-like squames seen and described in most cases. The curved white edges of exfoliation, compared by Mr. Wilson to frills, to scale armour, and to the ribbed sand on the shore, are well marked. The skin is not very painful, and less irritable than one would suppose. The colour is a full, bright, "inflammatory" redness, without lividity or the slightest pigmentation. The various organs appear to be normal, and the urine is free from albumen. The patient says that her general health is very good, except when the eruption has receded, as it did between the ages of sixteen and twenty-eight. No treatment had a decisive effect, and she left the hospital in May much as she came in.

CASE 2.—General exfoliative dermatitis, with free eczematous secretion; acute course; albuminuria; recovery.—A woman, æt. 26, was transferred to my care by Dr. Galabin in October, 1878, with general acute dermatitis. The following is a summary of the case:

Ann S—, suffering from pelvic cellulitis, was attacked while in the ward with a papular, measles-like rash on the trunk and legs, accompanied with light sore-throat and raised temperature. Two days later, the temperature rose to 104°, and albumen appeared in the urine. The rash was not like that of scarlatina, measles, or rubella; nor was the state of the tongue, throat, or other organs, like that in any of those exanthema. It had spread over the whole body excepting the palms and soles in a week, vesicles appeared on the chest, bulks, on the arms and legs, and a raw weeping patch behind one ear. Three weeks from the first appearance of the rash, the fever and albuminuria had disappeared, and free desquamation was going on; branny, like pityriasis, on the scalp and face, in large flakes on the hands. Impetiginous crusts had formed on the chin and on the limbs, and there was profuse serous secretion from the ears, neck, and arms. As involution went on, the previous pain was succeeded by intolerable itching, which gradually also disappeared. An abscess formed in one axilla; and a fresh papular eruption appeared on the chest and limbs on

the 6th of December, which only lasted three days. The patient was discharged perfectly well on the last day of the year.

This case resembles that of Dr. Wilks' referred to above (p. 262), and also one which I published in the twenty-third volume of this Series (1877). That was an acute universal, vesicular, and weeping, but also desquamative, dermatitis with pyrexia, occurring in a patient who was the subject of chronic tubular nephritis. It ran an acute course and proved fatal in four days.

CASE 3.—*General exfoliative dermatitis, beginning as recurrent eczema; profuse desquamation; weeping; acute course; albuminuria; recovery.*—Ellen D—, æt. 42, was admitted under my care into the clinical ward in July, 1879, presenting the characteristic features of universal exfoliative dermatitis. I may add that Dr. Baxter was so kind as to come down to see this case, and entirely agreed in the diagnosis. Her account was that in her two last pregnancies she had suffered from a slight eruption on the feet, and that this had spread during the last three years to the rest of the body. The whole of the surface (except the scalp and face), both trunk and limbs, including the palms and soles, was covered with a dusky red, scaly eruption. There were a few large scattered vesicles and some petechiæ. Here and there moist weeping spots were to be found; but the surface was generally dry. The scales were large, thin, not imbricated and not adherent, and were continually shed in great flakes which filled the bed. There was moderate pyrexia; and not only albumen in the urine, but also pus and mucus with other symptoms of cystitis. For a time she was exceedingly ill, but gradually and slowly improved, the scales became less abundant, the redness disappeared, the urine became normal, and she went out well at the end of August.

There was then only slight ordinary "dry scaly eczema" on the upper limbs.

She came to me during the following October and November with one spot of still persistent dermatitis on the flexor aspect of the left forearm just above the wrist. This presented no characteristic features except its obstinacy, and the fact that it produced superficial ulceration, so that slight scars remained after she was finally cured.

The following cases show that the same anatomical condition which is seen in general desquamative dermatitis or "*Eczema foliaceum universale*" (as distinguished from the perfectly dry cases of "*pityriasis rubra*") may also occur as a local affection.

I. A big florid woman of 44, came among my out-patients with somewhat extensive inflammation of the skin. It had lasted for two years. She had not before suffered from anything like it, and said that she was always perfectly well. There was no evidence of gout. Both legs were covered with large, thin, coherent flakes of epidermis, yellowish-white in colour, not closely adherent to the skin beneath, and when removed, leaving a dry, red, somewhat tender surface, like that of psoriasis. The affection was not confined to the flexor or extensor surface, covered both hams and extended in a less severe degree to the thighs, nates, and abdomen. On the outside of both arms a similar condition was observed, but it was less marked, and occurred in patches instead of being continuous. There was not much itching or pain, there had never been any weeping, and the skin was not infiltrated. A week later, after the application of an ointment containing two drachms of *Liquor Carbonis Detergens* to an ounce of lard, there was active ordinary exudative dermatitis set up (traumatic eczema). A superficial ulcer formed on one leg below the calf and there was considerable pain and disturbance. Gradually the acute symptoms thus produced subsided, and under treatment by alkalies and saline laxatives, with lead and zinc ointment externally, the skin recovered. After two or three month's observation, the patient was so nearly well that she ceased to attend. The treatment I here adopted had a more severe effect than I intended, but it appears to have acted as we often see stimulant applications prove useful; by substituting a more acute ordinary traumatic inflammation for the previous condition and so leading to the removal of the original disease.

II. An old man of 76, somewhat pale and thin, but in good health for his age, appeared with a chronic inflammation of both legs, which at the first glance looked like eczema. But it was strictly limited to the parts below the knee, it involved the whole of both feet except the soles, where the skin was hard and thick; it was dry, and the surface was covered with thin epidermic flakes as large as a crown piece and larger. On

removing them a very scanty, thick, tenacious secretion was found adhering to their deep surface, quite unlike eczematous fluid. There were no fissures, no ulcers, no varicose veins; and the skin was uniformly affected from a sharp line just below each knee downwards. The arms, trunk, and other parts were perfectly free from any lesion. There was considerable itching but no other constitutional disturbance. The affection had lasted two months. He gradually improved under local treatment chiefly with Ung. Plumb. Carb., and after several weeks was discharged with only a slight ordinary dry dermatitis.

III. A little pale woman of 40 came with a very simple "exfoliative dermatitis" of one leg. She had been under my care two years before for psoriasis, which was cured and had not returned. The anatomical condition closely resembled that last described, but the case was as much less severe than No. II as that was than No. I. The patient disappeared from observation before the effect of treatment could be ascertained.

IV. Hannah C—, æt. 41, has attended me on several occasions in 1879 and 1880 with large patches of inflamed skin on both legs above the ankle. The surface is red, angry, very irritable, and covered with a scanty, thick, white secretion, more like mucus than pus in appearance. This is seen when the large flat scales which conceal it are removed. These are not scabs, as in eczema, but epidermic scales, much larger and thinner than in psoriasis. Under the microscope they consist entirely of epithelium, with scanty pus-corpuscles on their under surface.¹ They are easily detached in flakes as large as a crown. This patient twice recovered under ordinary local treatment.

The following two remarkable cases are in accordance with what Dr. Baxter has observed, that a condition regarded by competent physicians as ordinary local psoriasis may assume the characters of a more serious and universal, dry, scaly disease, *Pityriasis rubra*. The hereditary character is also worthy of note.

Ellen P—, æt. 17, a stunted girl, looking four or five years younger, is still under my care in Miriam Ward. She has been more than once in the hospital under Dr. Wilks and Dr. Moxon,² sometimes for the disease of her skin, sometimes for

¹ See Dr. Liveing's 'Handbook,' p. 121.

² It is this patient to whom Dr. Moxon refers in his Croonian Lectures.

epilepsy, to which she has been subject from the age of ten. Once, also, she was attacked with acute pleuro-pneumonia, from which she completely recovered.

Her father, brothers, and sisters have healthy skins, but her mother has long been subject to a disease like that of her daughter (see the following case).

She was healthy when born, but when eighteen months old inflammation appeared behind the ears. This soon departed, but the following year returned and spread to the head and neck. Since then she has again and again recovered, and again and again the disease has returned with greater obstinacy. For several years it has been almost universal and constant. In February, 1879, when under Dr. Moxon's care with severe epileptic fits, the whole body was covered with a dry, scaly eruption, there was moderate pyrexia, but no albuminuria, and she went out much improved.

When she came under my care last April, the whole body was covered with a dry scaly eruption—scalp, face, trunk, and limbs, including the palms and soles. The skin was red, but there was no moisture, and scarcely any fissures, nor were the integuments infiltrated or thickened except by the masses of epidermis. Those on the scalp were small, and mixed with sebum; on the face, back, and trunk generally, they were comparatively scanty, the scales being thin, small, and readily detached, a true pityriasis. On the limbs this branny desquamation was replaced by a thick encasement of scales, covering the knuckles and fingers and toes, as well as the elbows and knees. The scales, however, differed from those of ordinary psoriasis in being more opaque, yellowish and dull, smaller and less adherent. The nails of both hands and feet were broken and deformed. There was no attempt at involution, and consequently no trace of an annular or gyrate form. The urine was normal. By a diligent use of baths and ointment constantly applied, the state of the skin was much improved; all the scales were removed, and the surface became apparently normal, except that it was still somewhat red and tender, and showed several pigment spots. She went out in this condition.

Sophia P—, æt. 48, the mother of the patient whose case has been just described, is now one of my out-patients. She also is the subject of a universal, dry, scaly disease, which I think

should be called *Pityriasis rubra*. Other good observers, however, consider it and her daughter's disease to be unusually severe and general psoriasis. The pathology of the two is certainly the same. In the mother's case there was no appearance of the disease until she was nearly forty. A scaly eruption then appeared on both forearms, and this gradually but quickly spread until it covered the whole of the body. At the present time there is not a sound spot anywhere. Scalp and face, palms and soles, every region is affected. The nails are brittle and deformed. There is not, and never has been, any exudation. The scales are generally numerous, small, loose, and not "pearly" in appearance. On the whole, they are not so thick as in her daughter. The face is almost free, being only red, smooth, and somewhat brawny, and the hands are in a similar state. Only on the limbs are the scales thickly massed, most so on the elbows, knees, and shins, but the distribution is much less regular than in ordinary cases of psoriasis. There are no patches or rings. The hair and teeth are fairly good. The tongue is clean and free from disease, the urine not albuminous. There is a great deal of irritation, the itching being sometimes intolerable. The patient is thin and weak, and there is slight pyrexia, but she eats well. There is chronic ophthalmia with great photophobia. Since the disease became universal it has never disappeared, but the condition of the skin is generally worse in the autumn. Ointments suit it best, especially vaseline. Arsenic she has found does her no good.

She has several children beside the one whose case has just been given, but no others of them have any affection of the skin. Her father's family were also free from any disease. Her mother, however, was subject to what she believes was the same disease—a general dry redness and scaliness of the skin—as long as she can remember. This, the only person affected in that generation of the family, died aged forty-eight. In the preceding generation the father (grandfather of Sophia and great grandfather of Ellen P—) was the subject of "the same complaint." He died an old man. Further back the family traditions do not go.

These two cases have considerable resemblance to Dr. Fagge's of acute general psoriasis proving fatal in a boy; indeed, the model of the face of that patient in our museum (No. 251)

would, I have no doubt, have been labelled pityriasis rubra by Devergie. The difficulty of diagnosis in this case and the two preceding ones is not between Pityriasis rubra and Eczema, but between Pityriasis rubra and Psoriasis. Indeed, the case of the daughter was called psoriasis by some of my colleagues in Guy's Hospital, and that of the mother was called psoriasis at an earlier period of the disease by Dr. Payne in St. Thomas's. In favour of this view are, I admit, the hereditary transmission and the occurrence of pigment spots in the girl. Nor can I doubt that the mother's case once presented the characters of ordinary psoriasis.

The objections to regarding either of the cases in their present state as psoriasis are, I submit, the universality of the eruption; the want of any selection of the favourite seats of psoriasis before it became universal, and of any special profusion in the same regions afterwards, together with the full and complete occupation of parts which it rarely affects; the absence of any attempt at a process of involution like that which almost always attends psoriasis, however inveterate; the presence of large loose squames in some parts and of branny desquamation in others, with the entire absence of the silvery, coherent and adherent, imbricated scales of psoriasis; lastly, the resistance to treatment, especially by arsenic.

Dr. Baxter would solve the difficulty by explaining my two cases as he does those of M. Guibout and of Dr. Fagge, to be psoriasis assuming the characters of, or developing into, Pityriasis rubra. But the cases are not similar. M. Guibout's patient had suffered from ordinary psoriasis for nine years. The universal deep redness with profuse exfoliation and severe constitutional symptoms which suddenly set in was clearly Pityriasis rubra in Devergie and Hebra's sense, notwithstanding that it received the odd designation of "*Herpétide maligne exfoliatrice*." It ran an acute course of six weeks, and then left the patient to his psoriasis. In Dr. Fagge's case there was no previous psoriasis; the disease, whatever it was, was the same throughout. In my two cases, even supposing that they were originally psoriasis, the gradual progress of the disease in an unusual, if not an unprecedented course, has at last completely changed its characters.

Dr. Baxter's hypothesis I take to be that eczema, psoriasis,

lichen, and pemphigus, show their pathological affinity by a common capacity of becoming universal and assuming new and unusually severe characters; but that, while thus converging, they in most cases preserve some mark of their origin. Thus eczema when generalised, becomes the moist, secreting form of universal exfoliative dermatitis (exemplified by Nos. 28 and 37 in the subjoined table). Psoriasis preserves its characteristic dryness and appears as Pityriasis rubra (in the restricted sense of Hebra, Nos. 3 and 4). Lichen develops into the severe general disease called Lichen rubra by Hebra. Lastly, Pemphigus assumes the well-known form described by Cazenave as Pemphigus chronique foliacé, of which he says: "L'éruption s'étend alors et peut prendre un caractère de généralité grave." To this last form would belong Devergie's two cases of Pityriasis rubra (Nos. 1 and 2), "qui se transformaient en pemphigus." Such a simplification is ingenious and attractive. But I do not think that it can be at present accepted. For (1) most of the recorded cases of exfoliative dermatitis have begun from none of these four local and ordinary diseases. (2) Others have arisen from erythema (No. 26), or from impetigo (No. 34), which no one, I believe, supposes to be "dartrous." (3) If we allow these four varieties of exfoliative dermatitis, may we not with little more extension of terms admit Hebra's prurigo as the generalised and inveterate development of infantile prurigo, or of the prurigo mitis of adults? (4) As I have already pointed out, universal dermatitis does not always assume the same characteristic anatomical features, and does not constantly affect the temperature, the urine, or the general health.

The difficulties of forming a judgment on the several questions raised concerning pityriasis rubra are extreme. I hope that this paper may contribute to a more extended knowledge of the facts upon which, when tested by future and larger experience, we may hope to found a sure and final judgment.

At present every fresh case which I have myself met with or which has been recorded by others, has added some fresh obstacle to making satisfactory generalisations. But I think a consideration of the evidence before us will lead us to admit the following statements as true.

1. The characteristic anatomical features of exfoliative

dermatitis present themselves under different clinical conditions. They may occur locally (p. 272) or universally; as a sudden acute attack having a short and favourable course (Nos. 32, 37); as a series of recurrent acute attacks (Nos. 4, 10, 11); or as a chronic disease from the beginning (Nos. 8, 24, 36).

2. The same disease, as judged by its redness, peculiar form of desquamation, and universality, may be perfectly dry throughout, or may be moist here and there in the flexures of the joints, or may secrete profusely.

3. Even when defined by terms made arbitrarily strait, a universal, dry, red exfoliative dermatitis may be accompanied by pyrexia or be free from it; it may produce severe emaciation, or leave the patient well-nourished; the kidneys may "sympathise" with the diseased integument, or the urine may remain healthy.

4. Instead of being confined, as Devergie thought, to persons who have passed the prime of life, subsequent observation has shown us that Pityriasis rubra may occur at almost every age.¹

5. Lastly, the grave prognosis of Devergie, made still more gloomy by the experience of Hebra, has been altered by the record of numerous cases which ended in complete recovery; and (what is more remarkable) of others in which the disease persisted incurable and unchanged for years but without seriously affecting the general health.

All we can at present do is to separate from one another such groups of cases as appear to be clinically and pathologically distinct, to look rather to these criteria than to anatomical variations in forming our groups, to bear in mind the practical objects of prognosis and treatment to which classification and nomenclature are a means, and to deviate as little as possible from the nomenclature most generally recognised in civilised countries, and especially from that of the greatest authority on the subject, the late Professor Hebra.

I would therefore separate as distinct from Pityriasis rubra the following forms of disease:

¹ The following are the ages of the patients affected with the more typical form of pityriasis rubra in the Table below:—Under 10, four cases, beside a fifth of pemphigus foliaceus (?) in an infant, which I have not included. Between 10 and 20, two cases. Between 20 and 40, seven cases. Between 40 and 60, ten cases. Above 60, five cases.

1. Acute universal dermatitis : a somewhat rare form of inflammation of the skin, superficial, *i.e.* affecting the Malpighian and papillary layers, so as not to leave scars, and ordinary, *i.e.* such as can be produced at will. It therefore resembles eczema in its anatomy, and runs through the same stages; first hyperæmia (the erythematous stage), then secretion (the weeping stage), usually assuming the appearance of *Eczema madidans* at once, but often showing vesicles, and occasionally papules or bullæ; lastly desquamation. Such cases are often called acute Eczema, but although idiopathic, superficial, and "common" dermatitis, they differ in their very rapid course, in their universal distribution, and in the absence of recurrence. Moreover, they are usually accompanied with pyrexia and general disturbance, and not unfrequently with albuminuria, approaching erysipelas in these respects. The prognosis is grave, but recovery is more frequent than death. The treatment is unlike that of eczema; it is essentially corroborant, quinine, stimulants, and mineral acids having been found most successful.

2. Cases in which Psoriasis becomes general and inveterate, but still preserves the characteristic form of the scales, the absence of exudation, except as the direct result of scratches or of cracks of the skin, and at least some trace of the predilection for certain regions. The successful treatment by arsenic in full and continuous doses shows the true pathology of such cases, but I cannot deny that severe and almost universal psoriasis may resist all treatment and lead to death.

3. Local exfoliative dermatitis, as I have described above (p. 272); differing from eczema in the abundance and size of the scales, in its sharp border, and its independence of the ordinary localisation; differing from Pityriasis rubra by not being universal.

4. Pemphigus foliaceus, as described by Cazenave, Hebra, and later writers. The few cases which I have seen of this remarkable disease occurred in women; they differed both anatomically and clinically from the exfoliative dermatitis as described by Wilson, and still more from the Pityriasis rubra of Hebra. One was certainly cured by an arsenical course of treatment.

I would—at least, for the present—classify as a single natural group of diseases, cases which conform to the descrip-

tions of Hebra and the three detailed cases of Mr. Wilson (Nos. 8, 9, and 10 in the table).

The most marked feature is their universal distribution, and also their rapid and irregular spreading, which is very unlike the gradual and, so to say, methodical extension of eczema.

The characteristic lesion is the production of large, thin, papery, or hop-like squames, unlike the silvery, imbricated, tenacious scales of psoriasis, the small, dirty, irregular scales of syphilis, the branny desquamation of measles or scarlatina, and the squamous stage of ordinary eczema. Their profusion and easy detachment are also remarkable.

In the most typical cases the skin is dry throughout, but sometimes a certain amount of secretion has been observed, apart from accidental cracks or injuries. This may be thick and gelatinous, or thin and malodorous, but it never has the stiffening property due to the richness in albumen of eczematous secretion, nor is it purulent.

The irritation is usually greater than in psoriasis, and the skin is redder and more burning.

In the more acute cases, possibly in all if they were observed at the outset, there is some pyrexia and general disturbance. Albuminuria is rare. If the disease becomes chronic (as is most frequently the case) these symptoms usually disappear.

The prognosis is not what Hebra supposed, and most of the cases recover; but when it has become chronic, pityriasis rubra is probably as incurable as ichthyosis.

The treatment is not satisfactory. Arsenic has failed in most cases to be of service. Even when the skin is perfectly dry, liquid applications, and especially warm baths, are much valued by many of the patients; but inunction with vaseline, or with lead or zinc ointment, made almost liquid by the addition of olive oil, is more commonly effectual. Tarry preparations are, I believe, ill borne.

TABLE OF CASES OF GENERAL EXFOLIATIVE DERMATITIS.

No.	Sex and age.	Origin.	Lesion.	Extent.	Constitutional symptoms.	Course.	Result.	Name given.	Author.
1	F., 61	Acute, idiopathic	Large scales, redness, exudation, bullae	Not universal; head, back, and arms free	Fever and diarrhoea	Intermittent	Recovery	P. rubra, ending in pemphigus	Devergie, Mal. de la Peau., p. 268, 1854.
2	M., 62	Gradual (?)	Desquamation, bullae	Not universal	" "	Chronic, 5 years	Improvement	" "	Ibid.
4, 5, 6	Sex and age not given	Gradual (?) idiopathic	Dry, deep red, scanty branny desquamation	Universal	Marasmus	Chronic, several years	Death	P. rubra	Hebra, Hautkrankheiten, i, p. 321, 1860.
3	M., 24 to 41	Acute, began with vesicular eczema of elbow	Dry, red, branny desquamation	"	Fever during each attack	3 mos., frequent attacks, each lasting several months, during 17 years	Recovery from last attack	" acute of Devergie	McGhie, Glas. Med. Journ., 1858, p. 431. Gairdner, Brit. Med. Journ., March 13, 1875.
7	M., 34	Acute, idiopathic	Redness and desquamation	"	Fever	6 weeks	Recovery	Acute general dermatitis	Wilks, Guy's Hosp. Rep., 1861.
8	M., 68	Acute, began with dermatitis of hands and feet; erysipelas	" "	"	Rapid pulse	Several months	Improvement	P. foliaceo rubra	E. Wilson, Dis. of the Skin, p. 177.
9	M., 69	Acute, idiopathic	" "	"	Absent	6 mos.; second attack, 18 mos. later, 3 months	Recovery from first attack; death from bronchitis during second	" "	Ibid., p. 186.
10	M., 50	Acute	Red, dry, desquamation	"	(?)	Repeated attacks	—	—	—
11	M., 55	"	Desquamation	"	(?)	" "	Recovery	—	Rayer, Mal. de la Peau. Cases cxvii, cxviii.

No.	Sex and age.	Origin.	Lesion.	Extent.	Constitutional symptoms.	Course.	Result.	Name given.	Author.
12	F., 13	"Same rash" on limbs a year before	Redness, with punctiform impaction; desquamation	Universal	Absent, slow pulses	5 months	Recovery	P. rubra	Hillier, Handbook of Skin Dis., p. 101.
13	F., 50	Gradual, idiopathic	Redness and desquamation	Palms and soles only	Absent	Several months	Death from lobular pneumonia	Eczema squamosum universale, seu P. rubra	Fagge, Guy's Hosp. Rep., 1867, p. 208.
14	M., 8	Gradual, idiopathic (previous rheumatism)	Redness, desquamation, rimae, profuse discharge	Feet only free	Severe (no albuminuria)	5 weeks	Death from bed-sores and exhaustion (adherent pericardium)	Acute psoriasis	Ibid.
15	M., 28	Acute, began with ordinary eczema	Dry, red, desquamation	Universal	Absent	4 months	Recovery	Exfoliative eczema	Wilson, Lect. on Eczema, 1870.
16	F., 9	—	Dry, red, branny desquamation	"	" (morbus cordis)	Several years	Not fatal	P. rubra of Hebra	Moore, St. Barth. Hosp. Rep., 1874.
17	F., 48	Gradual (previous rheumatism)	Redness, desquamation, pustules	"	Pyrexia	—	Recovery	General exfoliative dermatitis (P. rubra)	Sparks, Brit. Med. Journ., Nov. 6, 1875.
18	M., 20	Acute, idiopathic	Redness, desquamation, slight exudation	"	" angina, and bedsores (no albumin.), temporary bruit	8 months	"	Dermatite exfoliative généralisée	Percheron, Etude sur la Dermatite, 1875.
19	M., 51	Acute, previous ordinary psoriasis persisting throughout	Dry, red, desquamation	"	Severe	6 weeks	" ; a second similar attack	—	Guibout, <i>ibid.</i>
20	M., 51	Gradual, psoriasis from 51, becoming general at 44	Dry, imbricated scales	Universal; began on elbows and knees	Absent	6 months	Recovery under arsenic	Psoriasis rubide	(Case under Hardy). Boggio, <i>Triduo de Doctorat</i> , 1875.
21	M., 64	Gradual, beginning with ordinary eczema	Red, dry, with slight moisture; profuse desquamation	Universal	Ophthalmoid; night sweats; no albumen, both, &c.	12 months	Death from acute pneumonia	Dermatite exfoliative généralisée	Blaoken, 1875; quoted by Dr. Massé.

No.	Sex and age.	Origin.	Lesion.	Extent.	Constitutional symptoms.	Course.	Result.	Name given.	Author.
37	F., 26	Acute, idiopathic	Red, vesicles and bullae, profuse desquamation	Universal	Pyrexia, albuminuria	2 months	Recovery	Exfoliative dermatitis	Pyre-Smith, Guy's Hosp. Rep., 1881, vol. xxv, p. 270. Ibid., p. 271.
38	F., 42	Gradual, previous local dermatitis during pregnancies	Red, dry, except a few vesicles, profuse desquamation	"	Pyrexia, albuminuria, cystitis	—	"	"	"
39	F., 48	Gradual, beginning as psoriasis on forearms (not elbows or knees)	Red, dry, profuse desquamation	"	Pyrexia, no albuminuria, ophthalmia	8 years	Unchanged	P. rubra	Ibid., p. 274.
40	F., 17	Gradual, beginning in ordinary eczema (?)	Red, dry, profuse desquamation, pigmentation	"	Doubtful, a part from severe epilepsy	From infancy	Improved	"	Ibid., p. 273.

ON THREE CASES
OF
"REDUCTION EN MASSE."

By N. DAVIES-COLLEY, M.C.

THERE are few conditions which cause more anxiety to the surgeon, or which tax more severely the resources of his skill, than the persistence of symptoms of strangulation after the return of a hernia by taxis with or without an operation. During the last ten years many such cases have occurred to me, and various have been the causes which have led to this condition. The most frequent has been the injured state of the intestine produced by taxis or long incarceration, and unfortunately, but little can be done under such circumstances except to administer opium and trust to the reparative powers of nature. In three cases, however, the continuance of symptoms of strangulation has been due to the condition which has been described as "Reduction en Masse." As there are some points in each of these cases which seem to me peculiar, and as in two of them fatal results followed, which might perhaps have been avoided by a fuller knowledge and a different mode of operating, I have thought it desirable to place them on record.

CASE 1.—John W—, æt. 28, a dresser of sheepskins, was admitted into Luke Ward, on November 24th, 1880. For the

last six or seven years he had had a rupture in the right groin, but it had never been large, and he had not worn a truss.

On the 23rd, at 6 a.m., while he was at work, the rupture descended, and gave him pain. He went home and to bed. At 12 o'clock he took some medicine, and vomited immediately after. At 2 p.m. the rupture was reduced by a medical man without the aid of chloroform. The pain, however, still continued, and he vomited two or three times in the course of the night. On his way to the hospital he brought up some green matter. When I saw him soon after his admission he did not appear to be in much distress. There had been no passage of fæces or flatus from the rectum since the descent of the rupture. There was a good deal of pain referred to the vicinity of the umbilicus. No swelling could be seen in the groin, but the parts about the right inguinal ring gave a greater sense of resistance to the finger than on the other side. I could readily pass my finger into the inguinal canal, and I thought that I could feel something convex and elastic at its upper end. Although his symptoms did not appear very urgent, I thought it advisable to explore the parts under an anæsthetic.

Accordingly ether was at once administered. When he was quite unconscious and the abdominal walls fully relaxed, I could feel a swelling deeply placed behind the internal abdominal ring, and above Poupart's ligament. I estimated that its dimensions were from side to side $1\frac{1}{2}$ inches, from above downwards $1\frac{1}{4}$, and from before backward an inch. I made an oblique incision parallel to Poupart's ligament with its centre upon the internal ring. After dividing the aponeurosis of the external oblique, and some unusually thick muscular fibres at the lower edge of the internal oblique, I came down upon a membranous expansion, upon which a few fibres of the cremaster could be traced. As soon as I had made a free incision into this, which I supposed to be the transversalis fascia, a hernial sac bulged forwards through the opening. On snipping through this a jet of serum came away. I then laid it freely open, and found that it contained from $2\frac{1}{4}$ to 3 inches of rather empty small intestine, of a purplish colour. As far as I could ascertain there was no laceration in any part of the wall of the sac, which was abnormally thickened. When I tried to insert my finger within the neck

of the sac, I found that the peritoneum gave way before it, and I had to get my dresser to hold the lower extremity of the sac firm before I could steady its neck sufficiently to divide it with the hernia knife. The bowel could now be returned readily. The operation was performed with the usual anti-septic precautions. In about three weeks the wound healed, and the patient went out quite well upon January 15th, 1881.

From the investigation I made before and during the operation, I came to the conclusion that the hernia was an acquired one and that the whole or the greater part of the sac had been forced by the taxis to leave the inguinal canal, in which it had been enclosed in the sleeve-like process of the infundibuliform fascia. Having passed up through the internal ring, it then lay behind it, covered by the transversalis fascia, which I had to divide before the sac was able to bulge forwards into its usual position. It would seem, therefore, that this case falls under the class described by Mr. Birkett in Holmes's '*System of Surgery*,' as "*Reduction en Masse* by Displacement." The chief peculiarity to be noticed is that this form of reduction *en masse* has been usually noticed in femoral and not in inguinal hernias.

The other two cases are examples of reduction *en masse* by laceration of the neck of the sac. They both occurred during the operation for a strangulated femoral hernia.

CASE 2.—Mary B—, aged 53, a married woman, who had had four children, was admitted under my care into Lydia Ward, on April 7th, 1873. For two years she had noticed a gradually increasing swelling in the left groin, but she had never worn a truss. Latterly she had suffered from a cough, which had caused an impulse in the upper part of her thigh. The day before her admission she awoke at 5 a.m. with a pain in her "stomach," and soon afterwards she began to vomit. A little later her bowels acted, but not since. The pain increased, and she noticed that the swelling was harder and larger than usual. A doctor was summoned, and he applied taxis without success.

On admission.—She was of a fairly healthy appearance, and not in much pain. There had been no vomiting for ten hours. That which she had brought up she described as "bilious

stuff." There was a swelling in her left groin the size of a hen's egg.

After applying taxis unsuccessfully, I operated upon her later in the evening under chloroform. The sac was opened. It contained some omentum, which was adherent to the neck of the sac on the left side and behind, together with four inches of intestine of a reddish-purple colour, and in parts rough with recent lymph. After incising the neck of the sac, I had considerable difficulty in returning the intestine. Each portion had to be pushed up with the finger, and it seemed as if the neck of the knuckle was adherent to the parts around. Feeling dissatisfied, I drew it all down again, and I could then feel something like a band round the neck of the knuckle. This constricting band I partly notched with the knife, and partly tore it apart with my fingers. The bowel was then easily returned. The operation was conducted antiseptically.

The next day she had a rapid small pulse, and was still vomiting.

On the second day, as the sickness continued and she was in a low condition, I put her under chloroform, and made an incision through the parietes a short distance above and parallel to Poupart's ligament. This was next joined by a vertical incision to the operation wound. In doing so, I had to divide Poupart's ligament and ligature the deep epigastric artery. After opening the peritoneum and lifting up a quantity of omentum which lay immediately beneath it, I found the loop of intestine which had been strangulated dark and congested and in places gangrenous. Moreover, the neck of the loop was much constricted and acutely bent backwards upon itself. On drawing the loop forward some adhesions gave way, and some gas and liquid fæces escaped from an oval opening in the neck. I stitched the edges of the opening to the sides of the wound, but the vomiting still went on and she gradually sank and died early the next day, nearly three days after the first operation.

At the post-mortem examination we found general, but not very severe, peritonitis. On passing my finger into the first wound, it entered a cavity above Poupart's ligament and external to the peritoneum. Unfortunately, I failed to note the exact boundaries of the cavity, but, from what I can now

recollect of the case, I believe that it was at the upper and anterior part of the pelvis, between the peritoneum and the pelvic fascia. Its walls were ragged and coated with blood and lymph. It would appear that in my first operation I had pushed the bowel into this space, and that when I had drawn it back again I had felt with my finger the opening which led from the artificial cavity into that of the peritoneum. Probably I had enlarged this opening with the hernia knife and my fingers, but I had failed to replace the intestine through it into the abdomen.

CASE 3.—Mary A. E—, aged 46, a married woman, was admitted into Charity Ward on May 22nd, 1875. For two years she had had a rupture in the right groin, but had not worn a truss. Occasionally it went back of itself, and she could always return it easily. Thirty hours before admission the hernia had come down. Her bowels had been moved the day before. She began to vomit on the morning of the day on which she was admitted. She was found to have a femoral hernia in the right groin, rather hard, of a nearly spherical shape, with a diameter of about an inch, and very movable. Chloroform was administered, and the house-surgeon operated under my superintendence. The sac was opened, and a knuckle of intestine as big as a large-sized cherry appeared. He next proceeded to insert his finger and to incise the neck of the sac with the hernia knife. He then said that the intestine was adherent all round the neck of the sac, and he had to separate it with his finger. The protruding intestine could now be slowly reduced, but the house-surgeon said that it was still adherent to the outer side of the neck of the sac. This seemed also to me to be the case when I had carefully explored the parts with my finger. Bearing in mind, however, the case which I have just related, I tried to draw the hernia down again into the sac, but was unable to do so. It was therefore with some misgiving that I desisted from the attempt, and applied the usual antiseptic dressing, in the hope that, as I could not find the knuckle of intestine, it was safe within the peritoneal cavity. At first her condition was no worse than that which often follows the administration of chloroform. There was some vomiting, but only of a little

watery matter. This gradually got worse, the pulse became rapid, and the abdomen swelled. Three days after the operation I divided the sutures, and, after reopening the wound, I explored it with my finger. It appeared to me that the femoral ring was quite clear, and that my finger could be passed through it into the abdomen; so I did nothing further. She died the next day.

I am indebted to Dr. Hilton Fagge for the following report of the condition of the parts at the examination which he made twenty hours after death.

On opening the abdomen the peritoneum was found to be generally inflamed. "A knuckle of intestine was seen to be still incarcerated in some pouch outside the peritoneal cavity; at the same time there was nothing in the sac. The latter, though easily invaginated into the peritoneal cavity, was in its proper place in the hernial protrusion space below Poupart's ligament. The piece of small intestine was next gently pulled out of the space now containing it, and it was found to be lying beneath the peritoneum behind the right ramus of the pubes, in contact with the obturator internus muscle. So that there had been a rupture, or the parts had been incised at the time of the operation at the inner part of the neck, and the bowel in passing up had got out of the peritoneal cavity. As helping towards this state of things, a thick cord ran from the origin of the iliacs on this side to the inner side of the neck of the sac, forming a somewhat projecting ridge beneath the peritoneum, and against which the returning bowel may very likely have lodged, and then turned under it down into the pelvis. This thick cord was the remnant of the foetal hypogastric artery on this side.¹

¹ *Note by Dr. Hilton Fagge.*—"With regard to the cord spoken of as tending to cause the subperitoneal reduction, a further examination showed that it came off from the internal iliac on the left side, that it ran along in, or rather beneath, the peritoneum inside the position of the vessels, and then getting to the abdominal wall was lost on it, where it certainly appeared to be running obliquely upwards to the median line, as the hypogastric artery might do. The cord contained a good-sized artery at its origin from the internal iliac, and this gave off branches to the peritoneum in the direction of the bladder, uterus, &c. (vesical and uterine branches?). About the middle it became much smaller, but continued on to the groin as a pervious vessel, where I lost it. It thus occupied much the course of the obturator artery, except that it did not dip down to the foramen, but was reflected on to the abdominal wall. The obturator artery and

"The piece of intestine, about two inches in length, was eighteen inches from the cæcum. It was of a dark port-wine colour, and the nipped portion had on the convex aspect of the bowel a number of small yellow points, where it was going to slough through, so that in a short time a part of the canal would have opened to the size of a sixpence. The neck of the false sac did not nip the bowel at all tightly; I should have thought with a grip insufficient to cause either obstruction or strangulation. I am also inclined to think that had there been any peristaltic action at all on the part of the intestine, it would have worked itself out of its incarcerated state. The bowel below the stricture to the cæcum was closely contracted."

Although I have heard of other cases similar to those which I have related, I am not aware that any writer has pointed out the risk of returning the intestine into an extra-peritoneal pouch after opening the sac in the operation for femoral hernia. It seemed to me, therefore, very important to place these cases on record, so that others may be aware of the possibility of such an occurrence. At the same time I would add a few words upon the conditions which predispose to it, the mode of its production, the way in which it may be avoided, and the treatment when it does occur. And, firstly, I consider that in these cases there is probably an unusual looseness in the connections of the peritoneum lining the femoral ring and the adjacent part of the abdominal cavity to the fibrous and other structures with which they are in contact. Perhaps, also, the neck of the sac lies more deeply than normal above the level of Gimbernat's ligament and the femoral ring. For example, in Case 3 it seems probable that the inner boundary of the neck of the sac was formed by the ridge of peritoneum which covered the obliterated hypogastric artery on its way to the anterior wall of the abdomen. If so, it must have been considerably deeper than the level of

the deep epigastric came off by a common trunk from the external iliac, the latter being quite normal, the former running over the hernial neck and down on its inner side to the obturator foramen, so that it occupied the surgically dangerous position described in anatomical works, but yet had escaped being wounded."

Gimbernat's ligament, the usual position of the neck of a femoral hernia. In the second place, the mode of production would appear to be as follows:—The operator divides the edge of Gimbernat's ligament on a plane superficial to the true neck of the sac, and having thus slightly notched the perineum at the femoral ring, he further widens the opening by thrusting in his finger. At the same time the narrow neck of the sac lying above the level of the femoral ring, and having escaped his knife, is pushed upwards, together with the adjacent peritoneum, which is readily separated from the abdominal wall. A cavity is thus formed, into which the knuckle of intestine is returned, while its neck remains in a state of strangulation as before. As the opening formed by the knife is on the inner side of the femoral ring, the false sac is naturally formed upon that side, and the fact that it lies within the cavity of the pelvis prevents any bulging which would betray its formation.

In order to avoid such an untoward event, I would suggest that the large flat director should be used in all cases in which the finger cannot be easily introduced into the femoral ring. We may be quite sure that this will pass into the interior of the peritoneal cavity, and the hernia knife when passed along its groove will be certain to divide the peritoneal neck of the sac as well as the more fibrous external constriction at the edge of the femoral ring. If there should now be any difficulty in returning the protruded intestine I should advise the careful division of all the structures which lie in front of the sac as far upwards as Poupart's ligament. This can do no harm, for it does not entail the interference with any important vessel or ligament, while it lays bare all that part of the hernia which lies outside the femoral ring, diminishes the depth which has to be traversed by the finger in order to reach the neck of the sac, and, lastly, by cutting through the attachment of the upper extremity of the falciform ligament to the under surface of Gimbernat's ligament, it may remove an obstacle to the return of the hernia. There is much reason for apprehension when a recently-descended femoral hernia does not slip up readily after the neck of the sac has been fully dilated by the incision of the hernia knife and the subsequent introduction of the finger. If the intes-

tine has, so to speak, to be packed up piece after piece by the finger, and if the operator has the sensation of dealing with a knuckle of intestine fixed by firm adhesions at the neck, although there is no appearance of old inflammation on the part which he can see; if, again, the cavity into which it can be pushed seems to be limited by adhesions, I think it is very probable that he is making a mistake similar to that which was fatal to the two patients whose histories I have related. Under these circumstances his first endeavour should be to draw the knuckle down again into the proper hernial sac. If he should be unable to do this, as happened to myself in Case 8, I consider that he had better make an incision above and parallel to Poupart's ligament, which will enable him to explore the inguinal portion of the abdominal cavity. Should it appear to be necessary, he might still further extend the opening by prolonging upwards the vertical incision to meet it, thus dividing Poupart's ligament and probably also the deep epigastric artery. I am disposed to think, however, that in most cases a free incision through the parietes above Poupart's ligament would be sufficient. The operator will then be able to examine the femoral ring from above, and it will be easy to withdraw the knuckle of the intestine from the extra-peritoneal pouch into which it has been thrust. Unless the mistake is at once rectified there is very little chance of saving the patient's life. The symptoms of strangulation continue, but, as would appear from my two cases, they are not so urgent as to make it obvious that another operation is immediately required. Probably the return of the hernia to the false sac diminished to some degree the constriction upon its neck. At any rate there was but little pain, and the vomiting was so slight as to render it doubtful whether it was not due to the administration of the chloroform. When it has become quite evident that an exploratory operation is imperatively called for, I fear that in most cases we shall find that some portion of the constricted gut has become gangrenous, and that all our efforts to save the patient's life by forming an artificial anus or otherwise will be unsuccessful.

CHRONIC BRIGHT'S DISEASE WITHOUT ALBUMINURIA.

A Thesis for the Degree of Bachelor of Medicine of the University of Cambridge,
read June 16th, 1881.

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THAT the urine in chronic Bright's disease is occasionally, or even not infrequently, free from albumen is by no means a novel observation. The object of this paper is to prove something more than that; it is to prove that in the earlier stages and in most cases even to their final stage, the urine of what is generally known as chronic Bright's disease with red granular kidney, is most commonly perfectly normal. More than this, its object is to prove, either that chronic Bright's disease is not a renal disease, although it frequently gives rise to a renal affection, or else that another disease must be recognised, which constantly precedes and prepares the way for Bright's disease, which may be called arterio-capillary fibrosis, or any other name that may be preferred to it. For my own part it seems preferable to retain the name of "*Bright's disease*" for the general condition, nearly all the pathological results of which Bright so accurately described, and to say that though the associated changes of granular kidney, hypertrophied heart, and atheromatous arteries were all described by him, yet his interpretation of their relations and causation was not in all

cases correct; that it is probably true in many cases of the acute disease, but that we have learnt in time so to extend our views that we now include a much wider field when we speak of the pathology of chronic Bright's disease than that which Bright described, though possibly not a wider one than he suspected when he insisted on the insidious character of the malady which bears his name.

It is not proposed to enter here into a minute inquiry and adjudication upon the merits of the varied descriptions of the histology of Bright's disease that have emanated during past years from Germany or other countries; that duty has lately been performed for us by a most judicial mind in Dr. Southey's recent Lumleian Lectures at the College of Physicians. Those observations, as they are thus ably presented to us in review, appear somewhat too minute; they are descriptions in many instances of small groups of individual cases rather than of a whole disease, and founded too much on pathological and too little on clinical observation; their detail is admirable, but their generalisations seem wanting. All the various writers seem to object most strongly to look for an explanation of what they see beyond the confines of the kidney, and in this Dr. Southey would appear to follow them.

Where there is so much confusion and disagreement it is difficult to say where we shall find a common standpoint and sure footing. I shall therefore speak of Bright's disease as I have known it by the traditions and doctrines of its birth-place, and as I have learnt it by my own observation. The traditional teaching concerning Bright's disease at Guy's Hospital cannot be better illustrated than by a quotation from Dr. Southey's lectures:—"In 1852, Dr. Wilks published an article, entitled Cases of Bright's disease, in the 'Guy's Hospital Reports,' 2nd series, vol. viii. He began by expressing his preference for the name of Bright's disease over that of Albuminuria; since renal disease, he found, might exist and no albumen escape in the urine, and albuminuria might occur temporarily without depending upon any such lesions as Bright had described in the kidney. Bright's disease, he acknowledged, was still very imperfectly understood, pathologically or clinically; but certain well observed structural changes in the kidney, accompanied by a regular train of

symptoms and secondary pathological phenomena, might be vouchsafed to indicate it.”—“At p. 238 he writes: ‘In chronic granular kidney the urine is often in good quantity, containing no deposits, and may or may not be albuminous.’ Dr. Wilks pronounces himself as a very positive dualist, and discriminates two principal forms of Bright’s disease—the large white and the small granular—and then describes the rarer or lesservarieties, the mixed forms, and secondary forms, of later writers.”—“He is the first author I have read who points out plainly that the large white kidney may have two modes of commencement: either by acute general dropsy, or quite insidiously. He is the first to speak of granular kidneys as indicating senility; to notice how (*vide* p. 256) ‘the older the patient is, the more liable is he to granular degeneration of the kidney;’ and he remarks upon the thick tortuous radial artery, the general anæmia, the characteristic pale urine, and the desire to micturate frequently, as well as the tendency to death by apoplexy or uræmia in this chronic malady. He compares the changes in the large white kidney to bronchitis in the lungs, which may be recovered from entirely, or may pass on, by extension of the inflammatory processes through the parenchymatous tissue of the organ, to degenerations analogous to those which take place in broncho-pneumonia. His description of Bright’s disease leaves nothing to correct and little to add; but he is not much captivated (*vide* p. 274) by Johnson’s theory of the mode in which the albumen escapes from the blood, attributing the albuminuria, straightforwardly, to obstruction of the circulation through the tufts and capillaries, and not to its transudation through the tubes, because of their being denuded of cells.”

Later on, after reviewing all the contributions to the pathology of the disease from that time (1852) to the present, Dr. Southey sums up:—“*Finally we must admit that the indefinite number of intermediate forms (asked for by Dr. Wilks in former times) between the two typical varieties are now admitted to exist in great number, and become an even stronger argument in favour of the doctrine of one Bright’s disease.*” In this remark I am most fully in accord with Dr. Southey. With regard to the next great advance in the pathology of Bright’s disease, which Guy’s

teaching has again offered to the medical world, let me once more quote Dr. Southey's impartial opinion upon a subject upon which I shall have to speak later. "Arterio-capillary fibrosis appears to us a change wholly apart from senile degeneration, and from atheroma; it is a most real change, separate from that muscular hypertrophy which usually accompanies it and involving the entire thickness of the capillaries and the walls of the small arterioles. Essentially, it is a chronic irritative or inflammatory change; it is the cause, perhaps, wholly of one form of renal degeneration; but it occurs in more or less degree in all. It is a change far more widely spread throughout the body than was at first suspected; it has a most important bearing upon those so-called accidents, secondary serous inflammations, liver cirrhosis, lung cedema, pneumonia, meningitis, cerebral and spinal complications, which were thought formerly to be due to the irritament of an impure blood. Its recognition is a great step in advance and one which will, in due time, be appreciated, but it is still probably a part only of the whole truth, which subtends the wide subject of chronic inflammation. But we still reserve our opinion that Bright's disease is a real entity, and that, while granular atrophy may be a consequence of this in its most gradual and complete degree, it is the renal disease which gives the characteristic features to the widespread general phenomena."

To proceed now more directly to the argument of my thesis, let us take up the subject where Dr. Wilks left it, and say that two forms of kidney are generally recognised under the term chronic Bright's disease, namely, that in which the kidney is more or less "large and white," this form being characterised during life by the presence of much albumen and much dropsy, and that in which the kidney is more or less contracted and red, characterised by the presence of little if any albumen or dropsy. With the former of these groups we shall have nothing to do in the present paper; it is unfortunate that they have ever been classified as chronic; they are strictly subacute; if they live, they recover, or become chronic, in either case changing their structure, so as to be no longer recognisable; if they die, they do so comparatively soon, too soon to be truly chronic, too late to be acute.

Now, in the various forms of Bright's disease most observers

are now agreed that the changes in the kidney may be arranged under three heads:—1. Epithelial changes. 2. Interstitial cell growth and fibro-hyaline change. 3. Vascular and perivascular thickenings. Of these the epithelial are associated more commonly with acute or subacute disease, and therefore with much albumen and dropsy; the interstitial with chronic disease, and therefore with little albumen and dropsy: but on the one hand the epithelial changes may frequently be met with in chronic disease, though alone it is more characteristic of acute; while on the other the interstitial changes are very commonly met with in acute disease, though alone they are more characteristic of chronic. The vascular and perivascular changes may be either chronic or acute, the acute vascular and perivascular changes comprising those described first by Klebs, afterwards by Axel Key, and others, and known as “glomerular nephritis;” the chronic vascular changes are the thickenings of Malpighian capsules, of arterial coats, and the fibro-hyaline exudations of Gull and Sutton, confirmed by Ewald and others. It is with the clinical history of these latter changes that it is proposed to deal at present. The fact that these anatomical changes should be found in the vascular structures itself suggests that its cause should be sought in the condition of the vascular system during life. Now, it is well known that the arterial pressure is greatly increased in all forms of Bright's disease, and we have therefore a ready explanation of the changes found after death. Increased work in resisting over-distension has produced hypertrophy of the muscular coat, while distension and strain have produced thickening of the fibrous and elastic coats; similar changes are seen in the intestine and other muscular and fibrous tubes when they have increased work thrown upon them. But if due to this cause not only should the arteries of the kidneys and the Malpighian capsules become thus thickened, but a similar thickening should extend to all the vessels of the body and to the adjacent tissues. This has been shown by the researches of Sir William Gull and Dr. Sutton to be the case; they have especially demonstrated it in the brain and spinal cord,¹ and have shown the extension of the fibro-hyaline thickening of the adventitia into the surrounding delicate nervous structures,

¹ ‘Trans. Path. Soc.,’ vol. xxviii, 1877.

Dr. Klein¹ has shown a very similar thickening occurring as an acute condition in scarlatina, in which disease I have elsewhere demonstrated that the arterial pressure is commonly unduly high.² Further evidence of the thickening of vessels produced by distension is seen in the well-known local thickening of vessels in an inflamed area; this also is well shown by Klein in the intestines and spleen of enteric fever. We should, therefore, be prepared to admit that in Bright's disease the vascular changes are sufficiently accounted for by the increased arterial pressure. The more sudden and intense the increase of pressure the more severe will these changes be, and the larger will be the amount of cellular exudation in the region of the vessels and in the capsules of the tufts.³

But if high pressure produces these changes in kidney disease there is no reason why, if high pressure occur without kidney disease, it should not produce similar changes. Now, I have elsewhere shown⁴ that high arterial pressure is not a consequence, but an antecedent of kidney disease. In the acute condition, as seen in scarlatina, the high pressure can be recognised in the pulse before (and experience has since shown long before) the kidney gives any sign of failure or albumen appears in the urine, and that treatment of this high pressure usually averts or cures the kidney trouble; while as a chronic state I have also shown⁵ that high arterial pressure occurs in some persons from youth upwards, apparently marking them out for future Bright's disease, and that it is common in lead poisoning, alcoholism, pregnancy, dyspepsia, and other conditions predisposing to Bright's disease; that it occurs in them long before there is any sign of renal failure or organic vascular changes, which probably require, in most cases, years to develop. It therefore follows that these chronic conditions of high arterial pressure will produce in the kidney and elsewhere the vascular

¹ Loc. cit.

² "Scarlatinal Convalescence," 'Practitioner,' 1875.

³ This is the reason for the extreme changes in so-called glomerular nephritis, but though this is common in scarlatina, it is not confined to this disease. I have seen it in non-scarlatinal cases. It only requires a sufficient intensity and suddenness of onset in a previously healthy kidney; there is nothing specific in it.

⁴ "Etiology of Bright's Disease," 'Trans. Med. and Chir. Soc.,' 1874.

⁵ "Clinical Aspects of Bright's Disease," 'Guy's Hosp. Reports,' 1879.

and perivascular changes of Bright's disease already referred to.

Again, since it has been demonstrated that this vascular condition frequently precedes and ushers in both acute and chronic *renal disease*, and that it produces the vascular changes characteristic of Bright's disease, it follows that this general and inclusive term, Bright's disease, indicates not so much a primary *renal* disease as a general or blood disease in which the kidney is especially liable to be attacked, though it is well known that it suffers not alone, but in company with several other organs, notably the lungs, which are almost constantly affected by bronchitis, the stomach and intestines, which suffer from catarrh, and the skin, which has catarrh of its sweat ducts.

Dr. Saundby, of Birmingham, in a most suggestive article,¹ entitled the "Functional Stage of Granular Kidney," has strongly supported the view I put forward in the 'Guy's Reports,' that the condition of permanent high arterial pressure is an early stage of granular kidney.

From these considerations it follows that we have to deal with three stages of chronic Bright's disease: first, *the functional stage*, which is limited to the condition of high arterial pressure without organic changes in either the vascular system or the kidneys; second, *the chronic Bright's disease without nephritis*, the stage of organic changes in the vascular system and in the kidney (for which, if thought desirable, the term "arterio-capillary fibrosis" might be employed); third, chronic Bright's disease with nephritis, the natural, but by no means the invariable, termination of the disease; epithelial changes have now taken place in the kidneys, or the cirrhotic changes are extreme, and the symptoms of renal disease have become prominent.

Of these stages the third is well known, it is the form of this disease commonly diagnosed; the second is the one to which this thesis is devoted. The first appears to have every probability in its favour, but it requires years to prove it, as the cases must be watched from youth into old age.

The kidney of the second stage, to the naked eye, is purely red, more or less granular, the capsules will be somewhat and

¹ 'Birmingham Medical Review.'

perhaps extremely adherent, the cortex atrophied little or much, the cut edge crenated, the arteries distinctly thickened, gaping, and prominent, the heart more or less hypertrophied; in some cases the kidney may look perfectly healthy, perhaps the arteries alone may look a little thick. The microscope in these cases will show thickened *membrana propria* of the tubules, thickened capsules of the Malpighian tufts, more or less intertubular fibro-hyaline thickening, the arteries thickened both by hypertrophy of the muscular and fibro-hyaline thickening of the intima and perhaps of the adventitia; the epithelium will be normal or only a little granular, not increased in quantity. These kidneys differ from those of the third stage, inasmuch as the latter to the naked eye show grey or yellowish granulations in the cortex, these appearances being due to excessive proliferation of the epithelium of the tubes; the condition is so distinct that it is easy to recognise, by the presence of grey or yellowish mottling, the existence of any epithelial changes in the kidneys. These latter kidneys almost invariably give rise to albuminuria, and not unfrequently to dropsy. These epithelial changes may probably come and go at any time in a kidney of the second stage, giving rise to the numerous exacerbations and intercurrent acute attacks to which these cases are so liable.

It is kidneys in the second stage, or red granular kidneys, which in my opinion give rise to no albumen in the urine nor any dropsy; they can be diagnosed by the cardio-vascular signs alone. In a series of papers, entitled "The Essential Symptoms of Chronic Bright's Disease,"¹ I first put forward this view, and if I appeared to do so with unpardonable dogmatism, it was because of the large number of cases which I had seen, though I failed then to record any. My chief argument at that time was founded on a table of 100 cases of granular kidneys which I extracted without selection from the pathological reports of Guy's; this table showed that 26 only, out of this 100, presented, during life, symptoms which would have been recognised as those of Bright's disease; the others came under treatment for symptoms of cerebral hæmorrhage, heart disease, lung disease, and sundry medical and surgical diseases; it was, moreover, found that these two

¹ 'Lancet,' vol. i, 1879.

classes of 26 cases with kidney symptoms and 74 without were also divided pathologically into exactly the same groups; the first 26 were cases of *mixed* or mottled granular kidney, the other 76 were *red* granular kidneys.

Dr. Saundby, in a short note on the "Occurrence of Dropsy in Granular Kidney,"¹ introduces a table showing the diagnosis—which was sent from the wards to the post-mortem room at the Birmingham General Hospital—in 98 cases, in all of which the kidneys were granular; out of these 98, only 22 were sent down with the diagnosis of Bright's disease; of the rest, 16 were diagnosed as "morbus cordis," 11 as apoplexy, 8 as lung disease, the rest as various medical and surgical affections, including 4 of erysipelas and 4 of gangrene. We shall be nearly correct, then, if we say that only about 25 per cent. of the cases of granular kidney give rise to the ordinary symptoms of Bright's disease, namely, albuminuria and dropsy, and we may add that these have *mixed* or *mottled*, not simple *red* granular kidneys.

The two plates which accompany this paper represent the condition of the kidneys in two cases in which the changes indicative of the red granular kidney, without inflammatory lesions (that is, with little, if any, small-celled growth), were present. For these excellent drawings, made to scale, I am indebted to my friend Mr. George Turner. In Plate I the changes are exhibited in a very well-marked degree. In Plate II the changes are slight; the kidney might be described as "coarse," indeed the existence of disease in this specimen has been questioned by some observers. Dr. Klein, however, has kindly examined the original slides, and states that he finds the following changes in both specimens, and that they are present in Plate I to a very advanced degree. "The *membrana propria* of the tubules is thickened, both in the cortex and in the medulla; it is accompanied by thickening of the hyaline capsule of the Malpighian corpuscles, involving the vessels of the glomerulus. There is a hyaline thickening of the intima of the arteries and thickening of the muscular coat of the smaller arterioles." Dr. Klein remarked that the changes were similar to those he has described as occurring in scarlatina, and in the intestines and spleen in enteric fever,

¹ 'Birmingham Med. Review,' April, 1881.

to which I have already alluded. Owing to the thickening of the *membrana propria* of the tubules the processes of that membrane which pass inwards between the cells, separating them from each other and retaining them in position, can be seen in Plate II more distinctly than in any specimen I have ever examined.

We may pass now to the consideration of the series of cases upon which I rely to prove my proposition that this stage of chronic Bright's disease gives rise to no symptoms of renal failure. These cases have been collected almost entirely from among the medical patients in Guy's Hospital during the brief period of two years 1879-1880, that is to say, out of a total number of about 4000 cases. They only include those in which there was what I considered positive evidence of organic disease. I have excluded many the apex of whose heart could not be detected, although they might have a very typical pulse; some too have been rejected whose urine has not been watched with sufficient frequency or whose reports are too imperfect to place any reliance upon them. None, therefore, have been included who presented the symptoms of high arterial pressure alone, without any evidence of hypertrophy of the heart or of renal disease.

The cases number sixty-one in all; of these twenty-one proved fatal while under observation and the results of the post-mortem examinations are appended. These examinations, be it observed, are not made by advocates of any particular theory, they are not likely therefore to be strained to suit the views here put forward. It would be difficult to find two more competent and impartial observers than Dr. Hilton Fagge and Dr. Goodhart.

In addition to their more prominent symptoms for which they sought relief, nearly all these cases presented the following characteristics which led to their diagnosis:—They all had the signs of high arterial pressure; they all had very considerable hypertrophy of the heart, those cases only being accepted in which the apex beat was in the nipple line or external to it; in many the arteries were tangibly thickened; in all cases the urine was free from albumen while they were under observation. In most of the cases it was altogether free; in eleven cases albumen was present on one or two rare occasions

during a long period of careful observations, this happened immediately after admission to hospital and during the time they were severely ill; in three cases, though absent during long periods of observation, it occurred just previous to death; in three other cases of typical chronic Bright's disease, the patients were admitted with albuminuria, which disappeared under treatment and they left without it. Three cases had urine very variable in its characters, sometimes albuminous, sometimes not. In the remaining forty-one cases albumen was never discovered in the urine. The exceptions to well-marked hypertrophy of the heart were seven in number—three of the fatal cases; in one, a case of phthisis, in which there was great wasting, the heart only weighed 11 oz., but the kidneys were very granular and the arteries thick; in another, a case of severe arterial disease, with much disease about the commencement of the aorta, the heart weighed only 12½ oz., the body was spare and the muscles small; in another case, of a female with much wasting, the heart weighed only 12 oz., the kidneys being, however, markedly granular and the vessels thick. In all the other cases the hearts varied in weight from 15 oz. to 30 oz. Of the cases which were not fatal, in *four* the hypertrophy was not proven by displacement of the apex beat; in one of these there was renal dropsy and occasional albuminuria, in another bronchitis and emphysema with severe epistaxis, the arterial pressure being extremely high and the arteries tortuous, hard and thick; the other two were ordinary and generally accepted cases of Bright's disease in which the albumen disappeared while under treatment. Of the twenty-one fatal cases all had thickening of the arteries visible to the naked eye; all had well-marked hypertrophy of the heart with the exception of the three cases mentioned above; in ten cases the kidneys were of the ordinary *red* wasted and granular variety. Two were large and granular, but proved on examination to have merely the vascular changes to be mentioned hereafter, with very little if any small-celled (inflammatory) growth. In three the kidneys were of the mixed or yellow granular variety. In five cases the kidneys appeared perfectly healthy to the naked eye, of good size, with smooth surfaces and thin capsules; in all of these, however, it is noted by Dr. Fagge and Dr. Goodhart (who made the

post mortems) that the small arteries were thickened and prominent; in three of these the microscope showed thickening of vessels, Malpighian capsules and stroma, the other two were unfortunately not examined microscopically.

To the reports of cases tables of daily observations on the urine are appended; in these tables are recorded daily observations on the quantity, specific gravity, solids, and albumen present in the urine, and in some also is added a record of the number of actions of the bowels in the same period. The columns of figures headed "solids" are obtained by multiplying the last two figures of the specific gravity by the number of ounces of urine passed; the product is a purely empirical number, without any relation to grains or grammes, but it affords a convenient standard for comparison, taking the normal quantity of urine to be 50 oz., and the normal specific gravity to be 1020, the normal solids will be $50 \times 20 = 1000$, a number which commends itself as a convenient standard. Unfortunately these tables cannot be relied upon as strictly accurate. The quantity of urine has been measured by the nurses and the tables constructed by the clinical clerks. It is always difficult to induce the patient to carefully save all the urine passed; this difficulty is more especially felt in the female wards. In many cases these tables can only therefore be taken as giving *minimum* quantities, and a liberal addition may often be made to them. Each table must be judged upon its merits in this respect, and it will not be found difficult to decide when they are inaccurate. This is especially evident when, without any apparent reason, very great variations occur from day to day in the quantity passed. The physicians to the hospital have been, as they always are, most liberal in according me free use of their cases; to them I tender my most sincere thanks. It is right that I should say they are in no way responsible for the diagnoses made in these cases, with many of which, possibly, they would not be disposed to agree; I have tried as far as possible merely to state facts and to leave others to draw their deductions from them. The main facts stated in all but two or three of these reports were verified by myself, all but these few exceptions having come under my own observation. Nearly all the observations on the pulse are from my own dictation.

I. Cases of heart failure.

The first group of cases consist of ten cases of failure of the heart due to high arterial pressure, of these no less than eight were fatal, and the diagnosis which was made during life was confirmed by the post-mortem examination. Such cases as these were described in my paper in the 'Guy's Reports' for 1879, and they need not now be discussed at length. Unfortunately, in these cases, as they were nearly all extremely ill, it was difficult or impossible to obtain a complete account of the urine. In the Case 1, however, a very valuable record was obtained, by which it is evident that the urine when he was first admitted was normal both in quantity and specific gravity, but that as his cardiac failure increased the urine, as it usually does under the circumstances of venous congestion, became scanty, but of very high specific gravity. It once contained a trace of albumen shortly after admission, and not again till the day before death; this is the more remarkable as severe venous congestion existed during the whole period. An accurate record of the urine has been kept in Cases 7 and 8. In Case 7 there was a trace of albumen on the day after admission, but it was never found again during her stay in the hospital of about three months. Her urine was, however, of rather low specific gravity, varying from 1010—1015. In Case 8 the urine was carefully recorded throughout the whole of her stay (nearly a month) and was perfectly normal in all respects.

In all the remaining cases albumen was entirely absent whenever the urine was examined, except in Case 9 in which it was usually present, but occasionally absent; this case is included only as showing that even with the mixed form of granular kidney albumen may not be present in the urine on all occasions.

In all of these cases there was well-marked (in some extreme) hypertrophy and dilatation of the heart, diagnosed during life by displacement of the apex beat, and in the eight fatal cases confirmed by post-mortem examination. In none of the cases was there any primary valvular lesion, although

the valves were thickened in several by the chronic hypertrophic endocarditis, which constantly affects valves subject to high pressure. In five of the cases there was severe aortitis deformans, giving rise to the bruit of aortic regurgitation in two (Cases 1 and 2), in both of which the valves were more or less shrunk and inefficient. These five cases (1, 2, 3, 4, 5) closely resemble Cases 9 and 10 of my previous paper. These cases of aortic disease, due to high pressure, can always be easily distinguished from those of true valvular disease by taking the pulse as a guide, as I have previously pointed out. The pulse of true valvular disease is essentially a pulse with starved, empty arteries; while in these cases, although the pulse may still have the character of "splash" more or less developed, it will be persistent, and the artery tortuous and usually thickened.

In Cases 6, 7, 8, 9, and 10, the mitral valve had failed owing to dilatation of the heart. In neither case was the valve diseased. In Case 7 the mitral regurgitant bruit was only of temporary duration. In Case 9, no bruit was heard, though regurgitation probably occurred; the cavities of the heart contained old ante-mortem thrombi. In Case 10, the ordinary bruit of mitral regurgitation was present, but her symptoms indicated great dilatation of the right side; her disease was complicated by perihepatitis and frequently recurring ascites; she closely resembled Cases 11 and 12 of my previous paper. In Cases 6 and 8 a presystolic bruit was heard; this was very well marked in Case 6, the bruit was typically presystolic. The post-mortem showed that the valve would admit four fingers and that it had allowed regurgitation to take place. In Case 8 the bruit was not constant, it disappeared as she improved. These cases resemble Case 6 in my previous paper, in which a presystolic bruit occurred with a normal mitral valve. I gave my reasons in that paper for accepting Dr. Turner's observations, that a presystolic murmur frequently occurs in dilated hearts with wide mitral orifices; that it is in fact due to a commencing regurgitation which is arrested; that the first sound as heard in these cases does not indicate the commencement of systole, but only the moment of closure of the mitral valves. Their closure may be delayed either by their rigidity, or by the dilated orifice, or by their shortened

chordæ, until an appreciable time after systole has commenced ; during this interval regurgitation takes places, it is cut short by their closure, and the bruit is arrested by the first sound, giving it the character of the presystolic bruit.

These cases of mitral failure can, like the others, be distinguished from true valvular disease by the pulse, which is persistent, while the artery may be more or less thickened. The persistent pulse appears to be very rare in ordinary mitral valvular disease, where again the arteries are rather starved than over-filled. These cases lose the characteristic quality of *length*, owing to the usually prolonged systole being cut short by the regurgitation through the mitral valve, giving the blood two modes of exit from the ventricle.

In all of these eight fatal cases the arteries were thickened and the kidneys more or less granular. Each of the cases is worthy of more careful study from its own distinctive characters, but time will not allow me to refer to these, which would, moreover, distract from the main object of this paper.

II. *Cases of lung failure.*

This group includes eleven cases, of which six terminated fatally, and in these the results of the necropsies are given. In all the remaining cases unmistakable hypertrophy of the heart, very remarkable increase of pressure, and in most an easily recognised thickening of the arteries, proved the existence of chronic Bright's disease. They are all cases which presented clinically the symptoms of various forms of lung disease, but in all of which, with the exception of one (Case 14, which I did not see), an examination of the heart and pulse enabled me to recognise the underlying Bright's disease. This group is scarcely sufficiently well represented to convey an idea of its importance. The association of bronchitis with various general, or so-called blood conditions, is too well recognised to need demonstration here ; its coincidence with the ordinary nephritis of Bright's disease has been frequently pointed out. Dr. Wilks often remarks that you never see Bright's disease without it. The gouty diathesis is held to account for many cases of chronic bronchitis, and I am con-

vinced that unrecognised chronic Bright's disease, without nephritis, accounts for many more. In a very large proportion of the cases of bronchitis admitted into Guy's I have noted the association of high arterial pressure with it; but most of them are unavailable for the present purpose for two reasons: among those that were discharged relieved it was difficult to demonstrate the organic changes of Bright's disease, because the emphysema of the lungs disguised the hypertrophy of the heart; of those that died, nearly all came in severely ill, and in this stage they mostly have a trace of albumen in the urine, whereas, if they had been seen before the severe symptoms arose, the urine would probably have been found normal. I have notes of three very striking fatal cases of this description, in which the symptoms were so entirely pulmonary that they were regarded clinically, two as cases of severe bronchitis, the other as one of pleurisy, but in each the exceedingly high arterial pressure led me to recognise Bright's disease. All of these three cases had a faint trace of albumen in their urine, not more than their venous congestion might well have accounted for had their kidneys been healthy, but still sufficient to exclude them from this paper on Bright's disease without albuminuria. Case 11 is a very typical example of this class: the patient was a chronic bronchitic, and liable to attacks of very great severity, when he became intensely cyanotic. He had previously been in the hospital, and was regarded as a case of bronchitis and emphysema; his urine was then normal. On admission on the present occasion he presented his usual symptoms and was deeply cyanosed. His urine was scanty and of very high specific gravity; it was free from albumen. The first time I saw him I noted that his pulse was "very persistent, long, and of extremely high pressure," and I attributed his condition to Bright's disease. After he had been in a week I noticed that the pulse, though still persistent, had become shorter, owing, as I supposed, to failure of his mitral under the high pressure; on this occasion I found that his first sound had come to resemble a presystolic bruit, "such as I have often heard in dilated hearts." After this he had attacks of faintness and his pulse became intermittent. Rather more than a fortnight after admission his urine was found to be albuminous, and he gradually sank

and died in a fortnight. If this patient had come in about a fortnight later than he did, he, like the rest, would have been excluded from this paper.

Case 12 was a very striking one, though the kidneys only weighed 5 oz. together, the urine was free from albumen up to the time of her death; unfortunately she was only a few days under observation.

Case 13 was very complicated, and was thought to be pulmonary. He developed jaundice while in the hospital, and his urine, at first normal, afterwards became slightly albuminous; the account of it is unfortunately scanty. His arterial pressure was recognised as high, and his heart as hypertrophied. He had very granular kidneys, a heart of 17 oz., and, in addition to his bronchitis, a gall-stone was found in the common duct.

Case 15 is noteworthy because he had renal dropsy and yet no albuminuria, a condition which is by no means infrequent, no less than twelve examples occurring in this series. He was twice under treatment in the hospital. The report contains a very full and accurate record of his urine. It contained a trace of albumen the day after his admission on the first occasion, which disappeared the following day; it again contained a trace on two successive days during his second stay in the hospital. The character of the pulse, the dilatation of the heart, the renal dropsy, associated with his bronchitis, make the diagnosis in this case undoubted, and it receives confirmation perhaps by the occurrence of albumen on these three occasions.

Cases 16, 17, and 18 are all ordinary cases of chronic bronchitis and emphysema, in each of which the heart was distinctly hypertrophied, the apex beating one inch, one and a half inches, and half an inch outside of nipple line in each case respectively, and the signs of high arterial pressure being unequivocal. The urine was very carefully watched and recorded in all three cases, and it was always perfectly normal.

Case 19 had previously been in the hospital with bronchitis and albuminuria; on this occasion, though having the signs of high arterial pressure well developed, and the pasty aspect of Bright's disease, his urine was perfectly normal, though rather excessive in amount.

Case 20 was a very complicated one, both clinically and

pathologically; perhaps it ought to have been excluded as being doubtful, though I cannot think that any doubt pertains to it. The fact that the heart was found to weigh 17 oz., without valvular disease, and that the arteries were recognised as thickened by the naked eye, would fairly prove the existence of the vascular changes of Bright's disease, whatever the appearance of the kidney might be. I thought that its sections showed the ordinary vascular changes, though but slightly marked, of thickened capsules, thickened arteries, and thickened intertubular tissue, together with an excess of epithelium, which might have been accounted for by the existence of emboli in the kidney and the congestion they would give rise to. Dr. Fagge cast some doubts upon the kidney changes; but the importance of these changes is a question of opinion, as it depends on the variation of the limit of the so-called *normal*. There were several old ante-mortem thrombi in the ventricle (this is the third case in this series in which they have been found); the condition of gangrene of his toes and lungs, and also perhaps the acute enteritis, which set up his peritonitis, may be accounted for by embolisms from this source.

Case 21 is that of a man (proved by the post mortem to be gouty) affected by tubercular phthisis at the age of sixty-five. He is said to have lost two brothers by the same disease. He had been in Guy's three years previously with severe bronchitis, affecting the right lung most; signs of consolidation were then present at the right apex; his urine was perfectly normal on that occasion, as on this. The diseased condition of his radial arteries, associated with high pressure, led to a suspicion of his kidneys, though no hypertrophy of the heart could be found; they were subsequently found to weigh 9 oz., to be very granular, and to contain many cysts. The heart only weighed 11 oz., and this leads me to remark upon the fallacy of expecting hypertrophy of the heart to be invariably present in all cases of Bright's disease. There cannot be the least doubt that an hypertrophied heart may atrophy, as it probably had done in the present case, and also that hypertrophy may be prevented by impaired nutrition. I have repeatedly noticed a similar coincidence of absence of heart hypertrophy in cases of Bright's disease in which nutrition

has been gravely interfered with, as, indeed, I suppose all observers have done. I hold, therefore, that under certain circumstances the absence of hypertrophy in cases of high arterial pressure must not be taken to prove either that the pressure is not or has not been high, or that organic changes have not been produced.

III. *Cases of cerebral disease.*

The nine cases forming the third group are examples of chronic Bright's disease associated with cerebral disease, due to the changes produced in the arteries of the brain. Of these only two were fatal; the remaining seven are all well-proved cases of Bright's disease; in all of them the urine was usually normal. As in the lung complications, fatal cases without albuminuria are not very common, and this for two reasons: either the cerebral hæmorrhage occurs during an exacerbation of high pressure coincident with a passing renal congestion which gives rise to albuminuria, or else the venous congestion, associated with the stertorous breathing of fatal cases, is sufficient to cause the temporary appearance of albumen.

Case 22 is a remarkable one; he died of cerebral hæmorrhage within twenty-four hours of his admittance; his kidneys were found to be healthy as far as the naked eye could judge; they weighed $15\frac{1}{2}$ oz. His heart, however, weighed 22 oz., while the valves were healthy except for the ordinary hypertrophic thickening. His aorta was fairly good, but all his peripheral vessels were excessively thickened, and the larger ones atheromatous; his must have been a case of extravagantly high pressure without albuminuria.

Case 23, another case of cerebral hæmorrhage, closely resembles the last. His urine was examined on several occasions and found to be healthy, till the last two days, when it became mingled with blood, the results of catheterisation. The heart weighed only $12\frac{1}{2}$ oz.; the valves were healthy, but there was very extensive and general arterial disease, both of large and small arteries, all of which were greatly thickened. The kidneys weighed 12 oz., and appeared healthy, but their arteries were conspicuously thickened.

Portions were put aside for microscopical examination, but were unfortunately thrown away by mistake.

Cases 24 and 27 were both old men with sudden attacks of hemiplegia, whose hearts were decidedly hypertrophied, vessels thickened, and arterial pressure manifestly high. The urine in each case was normal, and a careful record of the urine of Case 24 is given, which extends over a long period; it shows a considerable variation in the quantity of solids excreted, and a deposit of uric acid crystals was frequently observed in it.

In Case 25 no hypertrophy of the heart was demonstrated, but his pulse was remarkably long, and the artery much thickened; he also was suffering from a sudden attack of hemiplegia, and his urine was albuminous for two days after admission; the albumen then disappeared and was seen no more during the six weeks he remained in hospital.

Case 26, another old man with similar symptoms; he had a striking family history; his father and one brother both died in apoplectic fits. His urine contained granular casts, but no albumen.

I had not an opportunity of seeing either of these cases myself, except Case 24; but the constancy with which sudden and permanent hemiplegia in old persons is found to be the result of hæmorrhage, together with their cardio-vascular phenomena, make the diagnoses tolerably certain.

Cases 28 and 29 are well-marked cases of cerebral softening; in the case of 28 the condition of the urine made the diagnosis too easy, for it frequently contained a trace of albumen, but in his case the arterial pressure seemed to have reached an extreme height, and was a much more constant guide than the urine, which on several days was free from albumen. In Case 29, also, the arterial pressure was exceedingly high, his heart was distinctly hypertrophied, and his urine of low specific gravity, though it never contained any albumen.

Case 30 was a patient on the verge of greater evil. She had lately become subject to severe attacks of vertigo, and was brought to the hospital in an unconscious state, having fallen down in one; she was unconscious two hours and then recovered without paralysis. Her heart was obviously hypertrophied, and her arterial pressure extremely high; her urine

was always normal. She suffered much from vertigo and headache, and it was not difficult to foretell that she would almost inevitably suffer from cerebral hæmorrhage at an early date.

The great importance of these cases is to be found in the value of these symptoms of high pressure in the prognosis and treatment of such cases. Dr. Broadbent has especially pointed out with what accuracy an attack of cerebral hæmorrhage can be foretold, and also how much can be done to ward off the evil day by appropriate treatment designed to reduce the high arterial pressure.

IV. Cases of renal dropsy without albuminuria.

This forms an unexpectedly large group, for it includes the nine cases in this section, and in addition Cases 15, 40 and 45. These twelve may be divided into two classes. The one group consists of some rare cases of chronic renal œdema, in which the material exuded into the tissues is of a semisolid gelatinous nature: these closely resemble the cases described as myxœdema, if, indeed, they are not still more intimately allied to it; and they appear to form a link between the ordinary serous œdema, and the chronic changes described as fibro-hyaline thickenings or exudations, upon the nature of which I think they throw very great light. The other group consists of cases of chronic Bright's disease without albuminuria, in which an exacerbation of these symptoms, which under ordinary circumstances would give rise to a transient albuminuria, produces instead a general œdema. The reason for this is tolerably obvious. The conditions giving rise to albuminuria and renal dropsy are no doubt closely allied; in fact, we have seen reason to believe that increased arterial pressure may determine either albuminuria or general œdema, or more commonly both. I have elsewhere pointed out,¹ that increase of arterial or venous pressure in the kidney produces albuminuria, while its reduction cures it. The same is known to be true of venous pressure in the production of dropsies, and it can also be demonstrated with regard to the arterial pressure.

¹ 'Trans. Roy. Med. and Chirurg. Soc.,' 1874; 'Practitioner,' 1876.

It is conceivable that, owing to local conditions the kidneys may be protected from an increase of arterial pressure, which is sufficient to produce general œdema; this might be effected by a contraction of the renal artery for the purpose of protecting the kidney from the strain of increased arterial pressure, a strain to which it is particularly subject, and which the microscope shows us produces terrible ravages in acute disease. There can be no reason why the renal artery should be denied such a power of protective contraction as this, which is accorded to all other arteries of equal calibre, indeed, it has been demonstrated to occur in the physiological laboratory by Dr. Lauder Brunton and Mr. Power in their researches on the action of digitalis. Another reason might be offered for the non-production of albuminuria in these cases, which perhaps explains its absence in so large a proportion of cases of chronic Bright's disease, namely, the thickening which has taken place in the vessels of the Malpighian tufts, through which no doubt it is difficult for the albumen to transude; and again, a third may be found in the thickening of the capsules, which must enable them to resist the expansion of the tufts and prevent their turgidity. Now, it is obviously easier for blood serum to transude through distended vessels than through comparatively contracted ones. In this resistance of the Malpighian capsules to distension, and the consequent strain thrown upon them in high arterial pressure, we have, no doubt, a ready explanation of their great thickening in Bright's disease; and in the resistance they ultimately offer when thickened and contracted, we may find a good and sufficient reason for the greater thickening and hypertrophy of the branches of the renal artery than those of any other artery in the body; it is undoubtedly a fact that thickening of the arteries is more constantly and frequently found in the kidneys than elsewhere. We have, then, three reasons to offer in explanation of the absence or small quantity of albumen in acute exacerbations of chronic Bright's disease.

1. Protective contraction of renal artery, especially true in acute disease.
2. Thickening of vessels of Malpighian tufts.
3. Thickening of capsules of tufts, preventing distension of vessels.

It is noteworthy, also, that a similar absence of albuminuria not unfrequently occurs in the renal dropsy of children after scarlatina; this happens in slight cases, so far as I have observed; they may possibly be explained by the protective contraction of the renal artery, or by the changes of the so-called glomerular nephritis. Of course it is impossible to accept the theory of the contraction of the renal artery unless one previously admits that Bright's disease is not a disease of the kidney so much as a general disease, and that the rise of arterial pressure is not due to local so much as to general causes.

But these speculations are comparatively idle. Let the cases tell their own tale. Case 31 is worthy of special notice. This man had a peculiar form of chronic general œdema, which was more solid and resistant to the touch than ordinary serous œdema, and was found after death to consist of semisolid, jelly-like material. It had not the chemical characters of myxœdema. I saw him when first admitted, and, from the condition of his pulse and the hypertrophy of the heart, had no hesitation in attributing his condition to chronic Bright's disease, although his urine was perfectly normal. This case was one, like others in this paper, in which the cardiac hypertrophy was altogether out of proportion to the kidney changes; the arteries generally were thickened.

Case 32 was one which appeared to closely resemble the last, but it did not prove fatal while under observation. In this case, however, the kidney changes were probably much more severe, for there was extensive retinitis and severe anæmia.

In Cases 33, 34, 35, 36, and 39, the attacks were acute, and resembled ordinary attacks of acute nephritis, but the albuminuria was absent; it was not completely so, however, in Case 35. In this one there was a slight trace of albumen for the first two days of his stay in hospital, and the dropsy and albuminuria went away almost together; it was followed by copious diuresis. All these cases might be perhaps open to some question, and this one most of all, as to whether they were subjects of chronic Bright's disease or only of primary acute attacks. They were all men over forty, and this alone, I think, is a strong argument against primary renal disease, which would appear to be rare at that age, unless the

result of some completely new poison introduced into the system, such as scarlatina.

In Case 35 the apex beat could not be distinguished, but the area of dulness was said to be enlarged. This is always an untrustworthy sign, and I am willing to abandon this case if his age, his bloated, unhealthy appearance, and his alcoholic excesses are not considered sufficient to give a fairly certain diagnosis of previous disease.

Case 36, again, was a most obscure case, and the apex beat could only be felt with difficulty, but the marked anæmia, the renal dropsy, the high arterial pressure, which is well demonstrated by the sphygmographic tracing, all combine to make a diagnosis of renal disease necessary.

In Cases 33, 34, and 39, the displacement of the apex was well marked, and the pulse highly characteristic. Case 33, moreover, had had one previous attack.

The remaining cases in this group (Cases 37, 38, 15, 40, and 45) were all cases of a subacute nature, in which the patients, having been more or less severely ill for some time with bronchitis and other troubles, gradually develop renal dropsy instead of simply cardiac. In Case 15 the urine only contained a trace of albumen on three occasions, though it was strictly watched over a long period. His appearance was very typical. Case 38 is the weakest of this series, for her cardiac hypertrophy was not absolutely demonstrated, but the diagnosis is considerably strengthened by the presence of albumen in her urine on three occasions. Case 37 had degenerate vessels. Cases 40 and 45, yet to be referred to, were both certain and very typical cases.

V. *Cases with gout.*

The association of Bright's disease with gout is too well known to need any notice here. In the six cases presented in this section, the existence of well-marked gout lends additional certainty to the diagnosis of these cases, in all of which the cardio-vascular lesions were well demonstrated clinically, although none of them terminated fatally. In all of them the urine has been watched very carefully, and the

observations fully recorded in tables. In only one was albumen ever present, and then only a trace on two occasions. The specific gravity was good in all the cases except Case 44, in which it varies from 1016—1010, though occasionally reaching 1020. The records of quantity I do not put forward as absolutely reliable; they are probably only approximate and can only be trusted as giving a minimum quantity. Case 45, already alluded to as having renal dropsy, had very great and easily detected hypertrophy of his heart; he came subsequently under observation as an out-patient, and though he had no albuminuria presented most typically the cardio-vascular signs of Bright's disease. Cases 40 and 41 are further strengthened by the existence of plumbism, another condition commonly associated with Bright's disease. I am disposed to think that we sometimes mistake cause for effect in this association. Is it not probable that in many cases it is not the plumbism which causes the Bright's disease, but rather the Bright's disease which causes the plumbism? This view well accords with the danger that certainly exists in Bright's disease, of poisoning people with the ordinary medicinal doses of drugs. Their excretory organs are usually inefficient, especially when clogged by catarrh.

VI. *Cases with severe epistaxis.*

The frequency with which severe epistaxis occurs in old people with high arterial pressure is very striking, and, for them, very fortunate; for, as I frequently have occasion to remark, "if their noses did not bleed their brains would." The symptom has been commonly observed as a precursor of apoplexy, and it is a fortunate accident which relieves arterial pressure and gives warning for further treatment. In these three cases the symptoms of Bright's disease are well marked. In Case 46 there were lumbar pain, a puffy face and the presence of albumen on two occasions, in addition to the cardio-vascular signs. Case 47 had degenerate vessels, and Case 48 had remarkably and persistently high arterial pressure, notwithstanding the hæmorrhage, which in most people would have produced very low pressure.

VII. *Cases with various medical and surgical diseases.*

In the following nine cases of various diseases the urine was known to be usually normal in eight; in the ninth no note of the urine was obtainable. In seven out of the nine cases hypertrophy of the heart and high arterial pressure were recognised during life; in one no note is made on the matter; in another, Case 50, the report was not available for reference at the time of writing. These cases point strongly to the importance of careful examination of the arterial pressure before any operation is attempted on a patient, though it does not appear that Bright's disease without nephritis is so dangerous an enemy to the surgeon as it is when nephritis is also present.

Case 49 was always suspected of Bright's disease, though it was never thought to be proved against him. The displacement of the apex was recognised and recorded; his urine contained albumen at one time, but he was undergoing catheterism, and the urine contained pus, which probably accounted for the albumen. The most remarkable feature in the case was the severe pericarditis, which failed to produce a bruit; the patient was examined by Dr. Goodhart a few days before death, and he only heard an indistinct first sound, although the whole pericardium was found to be covered with shaggy lymph.

Case 50 is of interest because the urine was repeatedly examined and found to be normal, so that no Bright's disease was suspected, but an attack of acute nephritis during convalescence proved fatal, and it was then found that chronic disease existed.

Of the next five cases (51, 52, 53, 54 and 55) all presented typically the cardio-vascular signs of organic disease, namely, displaced apex beat and high arterial pressure. In Cases 51, 52, and 53 the urine was always normal; Case 51 was diagnosed during life and proved by post-mortem examination. The heart hypertrophy was very well marked in the other two cases.

In Case 54 the arterial pressure was so high that I was led to remark that I was convinced that he had Bright's disease, and if he was exposed to a chill his urine would very

probably become albuminous. It happened soon after that he went down into "the park" on a very cold day, and the following day albumen was found in his urine; it only remained present for three days, and then entirely disappeared.

Case 55 also confirmed the diagnosis by once presenting a trace of albumen, but the report is not a very satisfactory one.

Case 56 is a very interesting one, though it lacks confirmation. It is the case of a woman who suffers from extremely severe attacks of vomiting and pyrosis, during which she can keep no food whatever in her stomach; these attacks last for variable periods, from a few days to a few weeks, and then pass off leaving her in her usual health. She had a pulse of extremely high pressure, a very unusual degree of it; her sphygmographic tracing shows its characters well. She was admitted into the hospital three times, and on the second occasion a diastolic bruit was distinctly audible in the aortic area; this faded somewhat while she was in, and at the time of her next admission I could not detect it. I believe in this case temporary aortic regurgitation occurred during an extreme elevation of pressure; it probably indicates a considerable degree of aortitis deformans. I have heard temporary aortic regurgitation take place in two other cases, and a short time ago some similar cases were reported from the Continent; it is probable that it not unfrequently takes place. I once had an opportunity of examining a case similar in many respects to the present one. I was asked to perform a post-mortem examination on a gentleman whose case had caused much perplexity to three or four of our most distinguished physicians; he had had attacks of vomiting, with intermediate returns to health; like the present patient, his urine had been repeatedly examined and pronounced normal. During a very severe and prolonged attack he died from exhaustion. Before the examination, after hearing the history I ventured to suggest chronic Bright's disease, and was told that this had been excluded by the condition of the urine. I found, however, typical red granular kidneys and a hypertrophied heart; this occurred five years ago.

Case 57 has been introduced here to prove a point. Although no record can be obtained of his urine, it may fairly be assumed to be normal, for his health was good

and his kidneys were practically healthy ; but his heart was nevertheless greatly hypertrophied, weighing $18\frac{1}{2}$ oz., although the valves were healthy. There was thickening of the arterioles of the pia mater, as well as of the arteries in the kidney; the kidney showed a little excess of stroma and some degeneration of Malpighian tufts. Dr. Fagge appends the note: "I think this case is distinctly opposed to the view that cardiac hypertrophy is secondary to an advanced degree of kidney disease." This case does not stand alone in this paper, nor is it even the best example of its class. In Cases 6, 22, 23, and 31, the kidneys presented very slight changes, and in all there was more or less hypertrophy of the heart; these organs weighed 23 oz., 22 oz., $12\frac{1}{2}$ oz., and 15 oz., respectively. Case 6 is a case of failure of the heart from high pressure, without valvular disease, and with general thickening of the arteries. Case 22 is a case of cerebral hæmorrhage also, with general arterial thickening. Case 23 was of the same nature, and Case 31 was the case of chronic renal oedema, with very slight kidney changes. These cases taken together almost conclusively prove the point, that there is such a thing as a disease with high arterial pressure and general vascular changes which is *not* secondary to disease of the kidney, but must be regarded as a general disorder. Inasmuch as we can trace the most gradual development of kidney changes in such cases, and we meet with every grade of kidney from the healthy to that of extreme disease, it is difficult to know where to draw the line that shall separate arterio-capillary fibrosis from Bright's disease, especially when we consider that these cases start with a certain functional disorder, and that their natural tendency is to go steadily, but surely, on to advanced kidney disease. I feel convinced that the more carefully the cases I have here collected are studied, the more certainly must it be impressed upon an impartial mind that we are here dealing with various phases of one common disease, and that its beginnings and endings are such as these cases indicate.

VIII.—Cases with well-marked albuminuria, which was variable or disappeared entirely.

This last group of cases I have introduced to make plain another point. So far, cases have been quoted to show that the *red* granular kidney does not give rise, under ordinary circumstances, to albuminuria. Not only has this symptom been occasionally absent, but it has been usually absent and only exceptionally present. I do not, however, wish to ignore the fact that a large proportion of our cases of granular kidney have albuminous urine, and that these were the cases which Bright recognised and described; I only desire to draw a line between the *red* and the *yellow*, or *mixed* granular kidney. Clinically, the *red* kidney has commonly normal urine, and the *yellow* kidney has commonly albuminous urine; while pathologically the *red* kidney has interstitial and vascular changes, while the *mixed* kidney has added to these epithelial changes. Now, it is the natural tendency of the *red* kidney to develop on small provocation tubular changes, and thus to become the *yellow* or *mixed*; it is these cases that are commonly recognised by the condition of the urine. If the attack is a slight one, the tubular changes may clear up again, as they do in scarlatina and other acute conditions; while if it be severe, the patient will go more or less rapidly down hill till he reaches death.

Cases 58 and 59 well illustrate the last remark. Case 58 is an undoubted case of chronic Bright's disease, for she has had two previous attacks of renal dropsy, and now comes under observation with the third; the first when she was fifteen, the second when twenty-eight, and she is now forty-three. The hypertrophy of her heart is not well marked, but her arterial pressure is exceedingly high, and although all her dropsy disappeared and her urine became normal, she can hardly be thought to have escaped with healthy kidneys. No doubt in her case the disease falls more on her kidneys as a local disease, and less on the system generally, than in most cases of the chronic disease. Supposing that she had much epithelial change in her kidney, as no doubt she had, the change from albuminuria with severe dropsy, to normal urine, was clearly

too quick for the epithelium to have recovered itself; it must then have still been much diseased while she was again passing normal urine; so that here probably we have a case of the mixed or yellow granular kidney in process of clearing up, and passing urine free from albumen. But this urine is not like that of most of our other cases; it was urine of low specific gravity (ranging latterly from 1012 to 1014), and answering to the description which is usually given of the urine of granular kidney which may be free from albumen, but is said to be of low specific gravity and of large quantity. This is the sort of case upon which such observations have been founded, and it is important to recognise the fact that when the specific gravity is low the kidneys are really seriously affected, that they belong to the *mixed* or *yellow* variety.

Case 59 shows a similar kidney going in the opposite direction, not towards recovery, but towards death; here there has been a temporary disappearance of albumen, but it returned again and was present when she was discharged. In this case the specific gravity was still lower, generally about 1009 or 1010, only once reaching 1015. In this case also, retinal hæmorrhages were present, a condition, as far as my experience goes, chiefly associated with chronic and severe kidney disease; I do not ever remember to have found them present in what I should call "chronic Bright's disease without nephritis."

Case 60 is another case with freedom from albumen, occurring in a case of manifestly bad kidney. The patient was sixty years of age, recovering from an attack of acute nephritis with dilatation of the heart; probably his cardiac condition, and the venous congestion it induced, accounted for the high specific gravity of his urine.

Case 61 shows a remission of albuminuria during an acute attack, and while the kidney was obviously much diseased. The presence of blood in the urine and the dropsy indicate that it was really a severe acute attack, yet in the course of his illness his urine would be free from albumen on one day, and bloody on the next. These relapses seemed to be determined by leaving his bed; while he stayed there he would do well; when he got about the albumen returned. I have remarked on this more at length in the record of his case. These cases

appear to indicate that the *mixed* granular kidney may pass non-albuminous urine, but that it will probably be of low specific gravity.

While describing the *red* granular kidney as often becoming the *mixed* or *yellow* variety, I do not wish to maintain that this is by any means the invariable course of events. I am only indicating one mode of termination of what I take to be the ordinary course of the most chronic form of Bright's disease. On the other hand, there are many other ways by which the *mixed* granular kidney can be reached. The disease may commence as an acute affection and afterwards become chronic, it may commence as chiefly epithelial and terminate as chiefly interstitial; again, it may be chronic and epithelial, or acute and interstitial. What has been cause in one case may be the result in another; thus general disorder may cause high arterial pressure, and this, in its turn, kidney changes; while, on the other hand, kidney changes may be primary and acute, and they may in their turn produce impurity of blood, and this general high pressure. But whether we read the tale backwards or forwards it is the same tale in the end, it is concerned with the same events, we only become acquainted with them in a different order; thus, we see chronic produce the acute disease or acute disease produce the chronic; if we know the beginning we can generally tell the ending if they live; for those which begin in the tubes end between them, and those which begin outside end within them.

CASES.

I. CASES OF HEART FAILURE.

CASE 1. *Hypertrophied heart; aortitis deformans; thick arteries; large granular kidneys; very rare trace of albumen; death.*—George C—, æt. 58. Dr. Wilks (Clin. clerk, Mr. Langridge). Admitted March 11th, 1879; discharged April 26th, 1879; readmitted July 16th, 1879; died September 17th, 1879. A horse-keeper, formerly a soldier, served through the Crimea.

No family history obtainable. Measles, scarlatina, and variola in childhood; syphilis twenty years ago. Had what he calls "rheumatic fever" in Turkey, but his joints were not swollen. Has drunk spirits freely. Six weeks before admission he became troubled with a feeling of fulness and soreness in the chest, his breath became short and laboured, and he suffered from cough. These symptoms lasted for three weeks, and then improved; they recurred a few days previous to admission.

On admission the patient was suffering from great dyspnoea with cough and bloody sputa, and great lividity of countenance. A quantity of blood was immediately abstracted from the left median basilic vein (about 18 oz.) with almost instantaneous relief to the patient, who was then propped up in bed in the sitting posture, this being the most comfortable to him. He is a powerfully-made man, with large square head and strongly-marked features. Well-marked subconjunctival cedema, but face not otherwise puffy. The report makes no mention of the presence or absence of dropsy. His face has the aspect of considerable distress from urgent orthopnoea and apnoea. He has a good deal of cough and the expectoration is now thick and uniformly tinged with blood. The breathing is chiefly abdominal, the chest somewhat barrel-shaped and uniformly resonant. There are mucous râles over the whole chest, most abundant on the right side. A small amount of albumen is present in the urine. On the day of admission I made the following note:—"Apex beat greatly displaced, two inches external and two and a half inches below nipple. To-and-fro bruit over aortic valves. *Pulse persistent. Vessels thick.* Pulse is now splashing, probably due in great measure to the bleeding. Chronic Bright's disease; aortitis deformans; perhaps some valvular disease; severe bronchitis." He presented typically the pulse of high pressure, and I frequently pointed him out as a good example of its results.

Unfortunately the report contains no further note concerning his urine during the whole period of his stay in hospital on this occasion. On March 17th the note runs:—"Is much better this morning, has not so much difficulty of breathing. The râles are not so many nor so loud on the right side of the chest; on the left there is slight tubular breathing." The correctness of the last observation is open to question.

April 3rd.—Patient usually wakes during the night with great pain in the epigastrium, nausea, but no sickness. Is obliged to get up and walk about the ward. The pain extends up into the right shoulder. On April 7th he is taking out-door exercise; on the 16th he has a slight relapse; on the 26th, having completely recovered from his bronchitis, he leaves the hospital; the double aortic bruit can still be heard at the base plainly.

On July 16th he is readmitted with a return of his former symptoms. A few days previously he had taken a fresh cold and had spat blood; he said that his hands were swollen, but there is no cedema of either hands or legs at time of readmission. He is anæmic; he is no longer described as cyanotic. The signs in his chest are much as formerly, but there is now some dulness at the base of both lungs behind, and moist sounds are more abundant over the back. The apex beat is stated by the clerk to be one and a half inches to the outer side, and two inches below nipple (half inch less in each direction than I formerly noted). Heart's action irregular. There is a double bruit heard over aortic valves, and a systolic bruit heard at apex which can also be heard in the axilla but not at the angle of the scapula. Area of cardiac dulness increased. Pulse, 80, persistent and irregular; it appears small and feeble when the arm is depressed, but when the arm is raised the pulse is full and suddenly collapses under the finger. The liver dulness is enlarged; lower edge of liver can be felt about two inches below the ribs; there is tenderness on pressure in the epigastric region. An account of the urine is tabulated below. July 19th.—Was obliged to get out of bed about 8 a.m. on account of difficulty of breathing; the attack lasted an hour and a half; has pain in the chest. Feet swollen this morning. During the rest of July and August he continues in much the same state, with increasingly severe pulmonary and cardiac dyspnoea; though his feet are much swollen, he had no cedema of his face or upper extremities. His urine continued free from albumen all the time. He suffered much from paroxysmal nocturnal dyspnoea, for which he was treated with morphia injections. Sept. 3rd.—Has had a very bad night. Dyspnoea extreme. This morning he has been expectorating mucus tinged with blood. Heart's action is very

excited. To-and-fro bruit very distinct at apex. Was bled to 4 oz. with instant relief. He only passed 10 oz. of urine, of high specific gravity, in the past twenty-four hours; this was increased in the following twenty-four hours to a pint. Sept. 10th.—His legs are so extremely oedematous that fluid is exuding from the distended skin. His breathing is becoming more difficult. The urine on standing stains the glass; there is a thick deposit, which on microscopical examination proves to contain phosphates, mucus, and *débris* of epithelium. Sept. 15th.—Dyspnœa is extreme. Pulse exceedingly weak and very irregular, so that one cannot count the beats. Legs have been pricked, and a quantity of fluid has escaped, but the skin is becoming of a mottled character, and patient has a worn and anxious expression. On this day his urine became albuminous. He died rather suddenly two days afterwards.

Date.	Quantity.	Sp. gr.	Solids.	Alb.
July 16	... Scanty	... 1028	... —	... A trace.
21	... "	... 1028	... —	... None.
28	... 56 oz.	... 1015	... 840	... A trace ?
29	... 50 "	... —	... —	... —
Aug. 1	... 88 "	... 1028	... 1064	... None.
6	... 80 "	... 1024	... 720	... "
9	... 20 "	... 1026	... 520	... "
11	... 10 "	... 1030	... 300	... "
15	... 10 "	... —	... —	... —
22	... 12 "	... 1030	... 860	... None.
26	... 12 "	... 1032	... 384	... "
29	... —	... 1033	... —	... —
Sept. 3	... 10 "	... —	... —	... —
4	... 20 "	... 1030	... 600	... —
5	... 20 "	... 1030	... 600	... None.
6	... 14 "	... 1030	... 420	... "
9	... 8 "	... —	... —	... —
10	... 10 "	... 1040	... 400	... None.
15	... 8 "	... 1025	... 200	... Albumen.

Post-mortem report (by Dr. G. F. Crooke, house-physician).—Face pale, jaundiced, lividity of lips, capillary injection of cheeks. A well-formed man, but considerably wasted. Oedema of feet, ankles, and thighs. Brain normal. Lungs, upper and middle lobes, crepitant, considerable emphysema

and bronchitis ; lower lobes tough, indurated, and oedematous. A good deal of muco-purulent secretion in bronchia. A small infarct in upper lobe and a large one in lower lobe of right lung. Pericardium somewhat thickened, containing a considerable quantity of serous fluid. A patch of recent pericarditis on left ventricle. Heart 27 oz. All cavities more or less dilated. Pulmonary and tricuspid valves normal. A widened mitral orifice, easily admitting four fingers ; valves generally thickened and contracted. Aorta studded throughout with calcareous spicules and plates ; valves thickened, puckered, incompetent ; likewise containing imbedded in their cusps calcareous spicules, especially down the centre of each cusp. Heart muscle (left) toughened from more or less fibroid induration. The *endocardium* thickened more than normal. Well-marked aortitis deformans, more particularly in arch and thoracic portion. Radials thickened and horny in places. Stomach and intestines congested. Liver 65 oz., shape normal ; localised patchy thickenings of capsule, with bands extending into parenchymatous tissue. Liver tissue firm, cuts toughly, shows the nutmeg characters fairly well. Spleen 5 oz., tough and small. Kidneys, 12 oz. Right presents a more congested appearance than left on section ; left presents a diffused yellowish colour, fatty changes more evident than right, both, however, were tough and firm, cutting with a rasp ; capsule removed with difficulty, leaving a torn surface. *The small arteries showed out plainly on section in both kidneys ; all seemed more or less thickened.*

Plate I represents a drawing from a section of this kidney ; the specimen shows great intertubular thickening of a fibro-hyaline nature, chiefly due to thickening of the basement membrane. The Malpighian capsules are much thickened. There is much hypertrophy of the muscular coat of the arteries and thickening of the intima and some of the adventitia. The epithelium of tubes, where it has not fallen out, is very granular and irregular, rather excessive in amount. No small-celled growth, indicative of inflammatory changes, is visible. This kidney shows typically the effects of prolonged high arterial pressure, with slight, and probably recent, catarrhal changes.

CASE 2. *Hypertrophied heart; aortitis deformans; aortic regurgitation; multiple aneurisms; thick vessels; slightly granular kidneys; urine normal; death.*—Richard K—, æt 48. Dr. Wilks (Clin. clerks, Mr. Scott and Mr. Garrard). December 8th, 1879—July 22nd, 1880. Family history unknown. Was a valet till twenty-eight years of age, after that a lighterman.

Has had no previous illnesses; no syphilis or rheumatism. His wife has had two children and no miscarriages. His illness commenced two years and a half ago, with a sudden pain in his chest coming on during a hard day's work, unloading a ship of wheat. During an effort he felt a sudden rick in his chest; he felt immediately very weak and faint, and had to sit down. He has never been free from pain since.

It is unnecessary for the present purpose to follow in detail the most excellent report that is given. Suffice it to say that he has suffered terribly and constantly from the most persistent angina pectoris I have ever witnessed. His life was only made possible by the relief he obtained from the constant use of nitrite of amyl; of this he had a large bottle, from which



he inhaled frequently. He had a paroxysmal cough, and a see-saw bruit in aortic area; the heart's impulse was in nipple line and four inches below it. At apex the systolic bruit was most marked. There were no physical signs of aneurism beyond deficient entry of air into right lung. His urine was clear, and of good quantity; sp. gr. 1015, acid, no albumen, no sugar. He was discharged on January 26th, and readmitted on July 7th, 1880. The angina was now worse than ever. He was propped up in bed, suffering constantly recurring paroxysms of pain. He had the typical aneurismal cough and loud stridulous breathing. He had some difficulty of swallowing. His pulse, which is unequal in the two

radials—the left being the fuller—is persistent and heaving in character, one evidently of high arterial pressure. Urine, sp. gr. 1018, clear, free from albumen. He steadily became worse, having several fits of choking, and died, after a struggle of terrible agony, on July 22nd.

Post-mortem (by Dr. Goodhart).—"Extreme emphysema of lungs:—a general disease, shown by silkiness, irregular lobulation of the lung, and pitting on pressure. The right middle lobe was solid, of a dark dull red, and over a solid ground were spread granular lobules, showing the bronchial origin of the pneumonia. The bronchial tubes were generally dilated, and full of thick gelatinous pus. There was no evidence now of any pressure upon the tubes by the aneurism. The lumen was not narrowed anywhere, nor was there any ulceration. Heart, 16 or 17 oz. Left ventricle much hypertrophied and dilated; the aortic valves had thick rounded edges, but were not very bad. The mitral had also a much thickened edge. The aorta was extensively diseased; it had numerous aneurismal pouches, which need not here be described; they extended all along the upper border of the arch from the ascending to the descending arch; there was also a considerable dilatation of the thoracic aorta. The sinuses of Valsalva were all thin, grey looking, and dilated into pouches. The kidneys were granular on the surface." Abdominal viscera all normal. *The vessels were very thick.*

CASE 8. *Hypertrophied heart with dilatation; aortitis deformans; mitral regurgitation; no valve disease; large granular kidneys; urine normal; death.*—Louis D—, æt. 51. Dr. Pavy (Clin. clerk, Mr. Hine). October 10th, 1878—January 7th, 1879. Readmitted May 4th, 1881. Died May 11th, 1881. His father is living; mother died in old age. He was a cook. He had an attack of rheumatic fever thirty years ago, but no serious illness since till ten months ago, when he was admitted into Guy's for bronchitis; this was about the beginning of 1878. After that time he was never well, constantly suffering from bronchitis and cardiac dyspnoea. He used to sit up in bed, suffering from much dyspnoea, and constantly complaining of the oppression on his chest and his want of breath. He had the expression of cardiac dis-

treas. His chest expanded very badly, and there was harsh bronchitic breathing, with occasional rhonchus and noisy prolonged expiration all over the chest; at the bases behind resonance was impaired, and there were some moist sounds. His cardiac impulse was masked by his emphysematous lung; the action was irregular; there was a systolic bruit to be heard at the apex. His pulse was very irregular, 66 per minute at the time of admission, and it was remarkably persistent, long, and incompressible. The temporal arteries were very prominent and tortuous, and his radials could be traced up the arm for some distance as an irregular cord. Urine, sp. gr. 1020, no albumen, no sugar; he often had a trouble to hold his water.

I registered the case on his first admission as one of chronic Bright's disease, with bronchitis, dilated heart and mitral regurgitation, and was constantly in the habit of pointing him out as a case of chronic Bright's disease without albuminuria, who showed in a typical manner the results of high arterial pressure; unfortunately, no further note occurs in this report about the condition of the urine, although I believe that it was frequently examined.



After he left the hospital on the second occasion he was for a long time an out-patient under Dr. Taylor, and I saw him on several occasions, his condition remaining the same. On his readmission on May 4th, 1881, his case was reported by Mr. H. E. Richardson, and his symptoms were as before, but much exaggerated and intensified; it was evident the end was very near. His dyspnoea was now extreme. The apex of the heart could be made out with much difficulty a quarter of an inch outside nipple line. There was a loud, blowing, systolic bruit at apex. Pulse 75, radial artery tortuous, easily moved about, hard; pulse incompressible. Liver is now somewhat enlarged. Urine a yellow colour and clear, with a white deposit at the bottom of the glass; sp. gr.

1020, alkaline, no albumen, no sugar. On boiling a white precipitate is thrown down, which clears up on adding an acid. The precipitate at the bottom of the glass also will not disappear on boiling, but clears up on adding an acid (phosphates).

May 7th.—Urine a deep yellow colour, sp. gr. 1022, no albumen, no sugar, no blood, contains a quantity of phosphates. A tracing of the pulse fails to demonstrate the high arterial pressure recognised by the finger, as the systolic expansion, which should be prolonged, is shortened by the existence of free mitral regurgitation. On May 7th he developed Cheyne-Stokes' respiration, which was observed on several occasions by the house-physician. On May 10th, the day before his death, his urine, which was of sp. gr. 1025, and very scanty, contained a trace of albumen for the first time.

Post-mortem (by Dr. Carrington).—Heart very large, weighing 27 ozs.; some fluid in pericardium. Hypertrophy and dilatation of both ventricles. All the valves quite healthy, except that there were one or two patches of atheroma on the anterior cusp of mitral valve. Cusps not thickened. A quantity of muco-purulent matter in the trachea, the cartilages of which were much ossified. Lungs emphysematous, and the tubes much dilated. There was a good deal of pus in the smaller tubes, especially of the right side; there was also general œdema of the lungs. There was pleuritic effusion on the left side and a small patch of lymph on the right. Lower lobe of right lung collapsed. The aorta and all the arteries were very atheromatous. Numerous dissecting aneurisms in aorta, into some of which a probe can be passed for a quarter of an inch. Kidneys large and coarse; they weighed together 20 oz.; they were granular and intensely congested (by mode of death); they appeared to belong to the large granular variety and likely to show much interstitial change. Arteries very much thickened. Liver congested and rather fatty. The arteries at the base of the brain were extremely atheromatous. There were some patches of softening in the brain.

CASE 4. *Hypertrophied and dilated heart; effusion into*

pericardium; *aortitis deformans*; *thick vessels*; *small granular kidneys*; *urine normal*; *death*.—William H—, æt. 65. Dr. Pavy (Clin. clerk, Mr. Bayer). August 2nd—August 13th, 1878; died. His father and mother both died between forty-five and fifty, cause unknown in both cases. He is a lighter-man, but has done no work for the last eighteen months. He has been a tolerably healthy man. He had typhoid fever in 1861. He has had jaundice once, if not twice. Has been a fairly sober man. He states that twelve months ago he had pleurisy, first of all on right side, then on left; after this he had inflammation of the lungs followed by bronchitis; whilst recovering from these attacks he noticed that his feet and ankles were swelling; this extended gradually to legs, thighs, external genitals, and finally to the abdomen; he has been under medical treatment for the five months previous to admission. He lies most at ease on his back. There is œdema in the parts mentioned and below his conjunctiva. No enlargement of liver or spleen. No cough or difficulty of breathing. Right side of chest hollow below clavicle and flattened; it moves less than left. Dulness on percussion over right lung, resonance fair over left; a few moist sounds at right base behind. Apex beat of heart not to be seen or felt. Heart sounds distant and rough; the heart sounds are more distinctly heard on right side of sternum than on left, as if the heart were drawn over. Radial pulse small and feeble, 120 per minute.

August 3rd.—Patient does not pass his water in large quantities; sp. gr. 1018, no albumen, no sugar. Temp. 98·5°; pulse 128. August 5th.—Twenty-eight ounces of urine during last twenty-four hours; sp. gr. 1018, no albumen, no sugar. August 7th.—His breathing is very difficult. Pulse 132 and very feeble. His abdomen has increased three quarters of an inch in circumference, measuring now forty-one and three quarter inches. August 9th.—Urine 28 oz., of sp. gr. 1016, no albumen, no sugar, reaction acid. August 12th.—Diarrhœa came on severely, he rapidly sank, and died the next day. No accurate account of the condition of the pulse is given in the report, but I find from my registration book that I diagnosed chronic Bright's disease at my first examination.

Post-mortem (by the house-physician).—"Much general

œdema. Right pleura firmly adherent all over chest wall, so that lungs could only be removed with difficulty. Fluid in the left pleural cavity. Lungs bulky, in parts emphysematous, rather firm, and very œdematous, frothy serous fluid exuding everywhere upon section. Larynx healthy, cartilages very much ossified. On opening chest, pericardium was seen to be distended with fluid, occupying a large portion of front of chest. Heart could be easily moved about in it. Sac contained 34 oz. of clear, straw-coloured fluid. Heart, 27 oz., enormously increased in size. Muscular fibre pale, soft, friable, and fatty. Coronary arteries rigid. Left auricle dilated, ventricle hypertrophied. Mitral valve healthy. Aortic valves acted well and did not allow reflux of blood, central valve had thickened margin about an eighth of an inch from upper surface, as if it had been bent on itself during life. Right ventricle much dilated. The whole of the ascending aorta and arch was much dilated and in an advanced state of atheromatous change, there being large calcareous plates in the wall of the artery. Peritoneal cavity contained a large quantity of ascitic fluid, surface of intestines dull and smooth, with lymph in places. Liver 59 oz., showing well-marked characteristics of nutmeg liver. Spleen 11 oz., firm and hard. Kidneys 9 oz., small; capsules adherent, surface granular. On section, cortex wasted, arteries rigid, and standing out on the section as easily defined round tubes."

The following case I ought perhaps to exclude from this paper; its value is very greatly diminished by the omission of the clinical clerk to record the result of each examination of the urine; its condition is only described at the time of admission. I feel confident that many other examinations were made, as I have it noted in my private note-book as a particularly striking case of Bright's disease without albuminuria.

CASE 5. Hypertrophied heart; aortitis deformans; thick vessels; small granular kidneys; urine normal; death.—Chas. R. E—, æt. 68. Dr. Wilks (Clin. clerk, Mr. P. James), April 24th—June 22nd, 1878. He is a French interpreter, has been a seaman. Has taken spirits freely, chiefly brandy. Had ague when a boy, and was subject to it until he was twenty.

one years old. Has never had rheumatic or scarlet fever. Twelve months ago went out of his mind and was in Wands-worth Asylum two months. For the last five months he has had a cough, with oppression on his chest and difficulty of breathing. On the morning of April 19th he noticed that his legs were much swollen when he got up. Hands and eyelids were slightly swollen. He is a tall, stout-looking man with grey hair; capillaries of cheeks congested. Great œdema of lower extremities, none elsewhere. He cannot remain long in the recumbent position without attacks of apnoea and fainting coming on; when he sits up to breathe the attacks pass off. No marked physical signs in lungs, though the breathing is hurried. Cardiac dulness increased. Apex to left of nipple and half an inch below it. Heart sounds irregular and tumultuous. Second sound appears dull at lower part of sternum. Pulse irregular and fast, 110. No pulsation of jugulars. Liver slightly enlarged. Spleen normal. No ascites. Urine acid, dark straw coloured, sp. gr. 1020, a mucous cloud, no albumen, sugar, or blood. April 25th.—Has frequent attacks of half fainting. Breathing hurried, but easier when body and chest are raised. April 30th.—I made the following note:—"There is a distinct, short, diastolic bruit heard over a very narrow area between the second costal cartilages; sounds elsewhere dull and similar in character. No thrill. Pulse *persistent* but splashing during systole." During the remainder of his illness his chief symptoms were oppression and pain in the chest, attacks of fainting, purpura of the legs, and delirium both at night and by day. His face became pale, and subsequently he had jaundice (May 28th), with irregularity of the pulse; and for the last month he passed his urine and fæces involuntarily.

Post-mortem (by Dr. Fagge).—"Brain wasted, membranes opaque, arteries atheromatous. Lungs very œdematous. Heart 30 oz.; mitral very good; aortic valves generally soft and flexible, but one had its edge rounded and thickened, and was to some extent shrunken. Heart's muscle good; aorta in extreme degree of aortitis deformans. Liver nutmegged, rather fatty; splenic capsule very opaque with plates of cartilaginous hardening; its tissue also hard. Kidneys very granular and extremely wasted, although their weight is set

down at 10 oz. Renal arteries almost choked by atheroma. No gout."

CASE 6. *Hypertrophied and dilated heart ; presystolic bruit ; no mitral disease ; kidneys very slightly granular ; arteries thick ; urine normal ; death.*—Richard M—, æt. 47. Dr. Pavy (Clin. clerk, Mr. E. A. Starling). April 30th, 1879. Died July 1st, 1879. A groom. Father has had "rheumatism." One brother died with swollen legs at the age of fifty-three. The patient had rheumatism twelve years ago. Has had a winter cough for some years ; has drunk freely both of beer and spirits. A month ago had a severe cold and cough, from which he got better ; a fortnight ago had a return of both ; six days ago he noticed his legs were swollen, and the day before admission his scrotum became œdematous. He is a well-made and well-nourished man, somewhat plethoric and heavy looking, cheeks red, and venules dilated and varicose. There is sub-conjunctival œdema ; the legs are swollen from the knees downwards, and the scrotum is œdematous. Liver one inch below ribs. Apex beat can be seen and felt one inch external to line of nipple and about three inches below it, in sixth space. Pre-cordial dulness increased. Systole laboured. A presystolic bruit is heard at apex and the second sound is reduplicated at base. Pulse full, persistent, vessels tortuous, very high arterial tension.



By Poud's sphygmograph. Pressure not recorded.

Chest large and barrel shaped, epigastric angle wide, expansion much impaired. Loud and noisy inspiration ; expiration prolonged and accompanied by râles on both sides. Urine rather dark, sp. gr. 1023, no albumen, bile, or sugar. A note of the urine was made on May 6th, 12th, 19th, 27th ; on neither day was albumen present. On May 27th he left the hospital, his bronchitis having cleared up a good deal ; his heart sounds remained the same. He was readmitted on June 17th. On this occasion he walked into the ward, but the exertion was

too much for him, and he became very blue ; his thighs, legs, feet, and scrotum are now very much swollen, the right leg measuring eighteen and a half, the left seventeen inches round the calf. Apex beats two inches external to nipple, and the area of cardiac dulness is largely increased ; there is a presystolic bruit and a reduplicated second sound. Both jugulars pulsate, filling from below. Very persistent pulse. Tortuous and distended temporals. Urine scanty, loaded with lithates, no albumen. Southey's tubes were subsequently introduced into his legs, but he became day by day more exhausted by his pulmonary and cardiac dyspnoea and died on July 1st.

Post-mortem report (by Dr. Fagge).—"Brain healthy. Lungs rather oedematous, some small apoplectic patches. Heart 23 oz., all the cavities dilated and hypertrophied ; aortic valves appear competent, but two were adherent half way to their centres and had very thick calcified edges ; the mitral orifice wide, admitting more than four fingers ; the edge of the valve seemed to turn in and there was rippling of the posterior wall of the auricle. Liver nutmegged. Stomach and intestines congested. Spleen indurated, 5 oz. Kidneys indurated, 11½ oz. Their capsules adherent, but I was not clear that there was any disease of importance ; *their arteries appeared rather thick.*"

CASE 7. *Hypertrophied and dilated heart ; temporary mitral regurgitation ; bronchitis ; albumen on one occasion ; high arterial pressure.*—Jane G—, æt. 57, Dr. Wilks (Clin. clerk, Mr. Rowlands). July 16th—October 11th, 1879. Family history good. Patient has always worked hard, has been much exposed to wet and cold. Is married, has had seven children. Has always been healthy up to seven years, when she had ulcers on her legs, which troubled her for some years afterwards. Two months ago she began to suffer from constipation and difficulty in passing water, which was scanty and high coloured. She has had pain in her left side and stomach, and shortness of breath ; oedema of feet and legs. She is a largely made, bloated-looking woman, with pendulous cheeks, subconjunctival oedema, and the aspect of cardiac distress. She has much dyspnoea. Vesicular murmur loud

and coarse all over chest; slight crepitations behind at the bases of the lungs. On admission pulse very irregular and a systolic bruit audible at the apex. A week later I note that "the bruit has disappeared, but the rhythm is still irregular; the second sound is accentuated and the apex beats one, to one and a half, inches external to nipple. The pulse is very persistent and many beats are long." The appetite is bad; bowels confined. The liver can be felt two inches or more below the ribs. The urine contained a trace of albumen on the day of admission, but never afterwards. It was generally of pale colour and of rather low specific gravity. For further details see table. August 2nd.—She is described as feeling better, cough less, heart more regular, no bruit. Liver still enlarged. Her condition continued much the same till the time of her discharge, October 11th, when it is noted that the bruit is still audible, and the feet swell at night time when she has been about all day.

Date.	Quantity.	Sp. gr.	Solids.	Alb.
July 17 ...	—	1012	—	A trace.
19 ...	—	1024	—	None.
21 ...	—	1021	—	"
26 ...	62 oz.	1012	744	"
28 ...	22 "	1012	264	"
31 ...	36 "	1015	540	"
Aug. 2 ...	—	1022	—	"
6 ...	—	1010	—	"
25 ...	—	1010	—	"
29 ...	—	1012	—	"
Sept. 1 ...	30 oz.	—	—	—
2 ...	40 "	—	—	—
3 ...	20 "	—	—	—
4 ...	50 "	1006	300	"
5 ...	40 "	1010	400	"
6 ...	44 "	1010	440	"
7 ...	26 "	—	—	"
8 ...	30 "	—	—	"
9 ...	38 "	—	—	"
16 ...	36 "	1012	432	"
17 ...	40 "	1010	400	"
18 ...	46 "	1010	460	"
19 ...	50 "	1010	500	"
20 ...	26 "	1015	390	"
21 ...	30 "	1015	450	"

340 *Chronic Bright's Disease without Albuminuria.*

Date.	Quantity.	Sp. gr.	Solids.	Alb.
Sept. 22	... 34 oz. ...	1010	... 340	... None.
23	... 40 „ ...	1015	... 600	... „
24	... 46 „ ...	1012	... 552	... „
25	... 30 „ ...	1012	... 360	... „
26	... 46 „ ...	1012	... 552	... „
27	... 36 „ ...	1015	... 540	... „
28	... 32 „ ...	1015	... 480	... „
29	... 50 „ ...	1012	... 600	... „
30	... 30 „ ...	1015	... 450	... „
Oct. 1	... 46 „ ...	1010	... 460	... „
2	... 30 „ ...	1012	... 450	... „
3	... 32 „ ...	1012	... 384	... „
4	... 34 „ ...	1012	... 408	... „
5	... 50 „ ...	1010	... 500	... „
6	... 46 „ ...	1010	... 460	... „
7	... 50 „ ...	1012	... 600	... „
8	... 40 „ ...	1012	... 480	... „
9	... 44 „ ...	1012	... 528	... „
10	... 40 „ ...	1012	... 480	... „

CASE 8. *Hypertrophied and dilated heart; presystolic bruit? very high arterial pressure; headache; urine normal.*
—Sarah B—, æt. 49. Dr. Moxon (Clin. clerk, Mr. A. P. Hills). May 3rd—May 27th, 1879. Book-keeper and manager of a hotel until eighteen months ago; since then she has followed no occupation. Mother died, æt. 65, of heart disease. One sister has had acute rheumatism, which was followed by heart disease. For last twenty-three years has been subject to headaches coming on about noon; at first they only occurred about once a month, last summer they became more frequent, occurring once a week, and now they sometimes continue for three days instead of one. When 25 years of age she had what she calls “rheumatic gout.” She now complains of constant pain over the back of the head on the right side; sometimes it spreads all over the head, and is so severe that she is afraid of losing her reason. A week before admission she had what appears to have been an attack of urticaria. She is a well-nourished woman, with a tranquil expression; venules of cheeks, chin, and nose dilated. Appetite bad, morning nausea, constipation. Abdominal viscera appear normal. Respiratory system normal. Cardiac impulse in fifth interspace external to nipple. A

presystolic bruit is stated to have been heard by myself and the house-physician. Radial pulse '84, large, long, incompressible, and regular; a pulse of very high tension. The



By Pond's sphygmograph. Pressure not recorded.

sphygmographic tracing confirms this description, and shows unusually high pressure. Sight very weak, cannot read a paper, sometimes even with very strong spectacles. (Unfortunately the report contains no account of the condition of her retina.) She passes a large quantity of urine; last summer it was as much as five or six pints a day, no albumen or sugar, sp. gr. 1012. Catamenia ceased eight months ago. An account of the urine is given in the accompanying table. Though frequently examined no albumen was ever discovered in it. Treated by the ordinary white mixture her headache was relieved, but not cured.

Date.	Quantity.	Sp. gr.	Solids.	Alb.
May 7	34 oz.	—	—	None.
8	32 „	1021	672	„
9	43 „	—	—	„
10	56 „	—	—	„
11	44 „	—	—	„
12	36 „	—	—	„
13	46 „	—	—	„
14	36 „	1010	360	„
15	52 „	1012	604	„
16	36 „	—	—	„
17	30 „	1018	540	„
18	44 „	—	—	„
19	60 „	1012	720	„
20	66 „	1010	660	„
21	80 „	1010	800	„
22	40 „	1020	800	„
23	50 „	1014	700	„
24	38 „	1012	456	„
25	43 „	—	—	„
26	74 „	1018	1332	„
27	38 „	1016	608	„

CASE 9. *Dilated heart with ante-mortem thrombi; softening of brain from embolism; old cerebral hæmorrhages; mixed kidneys; albumen variable, sometimes absent; death.*—Ann S—, æt. 43. Dr. Habershon (Clin. clerk, Mr. Morse). February 24th—March 31st, 1879. Her father is said to have had rheumatism, otherwise her family history is good. She states that she has had erysipelas five times all over her head. Fifteen years ago she first noticed pain in her heart and palpitation after dragging heavy weights. Since then she has often had winter cough, which has been especially troublesome these last three winters. She has kept her bed, more or less, on account of her cough, since last November. On admission she sits up in bed, is unable to lie on her back, face very blue and congested, looks very distressed, and has great difficulty in breathing. Appetite bad. Nausea. Bowels confined. Liver enlarged, three inches below ribs. There are signs in her chest of considerable bronchitis. Heart's impulse diffused, right side enlarged. Both sounds indistinct. No bruit. Pulse persistent, small, and long; high arterial pressure. Urine sp. gr. 1020, neutral, deficient in quantity, a great deal of albumen; no sugar. March 1st.—About 9 a.m. she had a kind of fit, but apparently without loss of consciousness. At 12.45 it was observed that she had partial loss of speech, chiefly answering "Yes" to all questions; there was loss of power in right forearm and hand, in right leg, and in muscles of right lower half of the face. There was loss of sensation all over the right side of the body. Optic discs normal, no difference between the two sides, some hypermetropia. The paralysis improved rapidly. March 5th.—Urine, sp. gr. 1026, only a trace of albumen. March 8th.—Urine contains a trace of albumen, sp. gr. 1023. March 10th.—Has recovered the use of her arm and leg, and is able to express her thoughts better; *no albumen in urine*. March 14th.—Patient is better; *no albumen in urine*. March 18th.—Patient looks very livid this morning, and her ideas seem very confused; it is supposed that she was seized with another fit in the night. She complains of great pain about the scrobiculus cordis. Breathing 41 per minute. March 19th.—Patient had another attack in the night, and was cupped on the shoulder; 4½ oz. of blood were drawn off. This morning

she looks very blue; resp. 46, pulse 120, temp. normal. She spits up a good deal more blood than before the attack. March 20th.—Urine dark coloured, sp. gr. 1025, quantity small, albumen about a quarter, no trace of blood. March 22nd.—Patient looks better. Urine sp. gr. 1025, a small quantity of albumen. She still spits dark liver-coloured sputa. She became worse on the 28th, gradually getting more blue and exhausted. She died on the 31st.

Post mortem (by Dr. Goodhart).—"Slight dropsy of legs. Body fat. Brain 43 oz. The vessels at the base were all of small calibre and thick, with patches of atheroma, which closed them in fast, so much so that I thought at first that the left internal carotid was plugged; it was not so, however, the canal was merely contracted, and after careful examination I could nowhere detect an actual plug in any vessel. The vessels under the microscope were decidedly thick. There was a large patch of softening affecting the grey matter of the convolutions of the left inferior parietal lobule, and extending irregularly into the white matter also, which occupied a considerable area behind and external to the central nuclei on this side" (the details of this need not be given here). "There was one patch of old brown extravasation in the uncinate gyrus, and another in the pons. Lungs somewhat decomposed; there was a large gangrenous cavity in the anterior part of the middle lobe, and around it several masses of pulmonary apoplexy. (The cavity probably took its origin from a similar condition.) Heart 25 oz; right auricular appendix full of large cystic ante-mortem coagula, their central parts being softened with grumous fluid; right ventricle thick and dilated; left ventricle also thick and very dilated, with, at the apex of the left ventricle, a half-inch sized mass of ante-mortem coagulum; the mitral a little stretched and thick; aortics also a little thick. The aorta had, just above the two anterior valves, a peculiar linear scar; the margins of the scar were sharp, its surface was depressed and thin, it tailed away into a point. I think it must have been due to some previous slit in the artery and to the healing of this. Aorta fairly good. Liver 75 oz., healthy. Spleen 6 oz., firm. Kidneys 15 oz; surfaces granular with a few cysts in them; the organs indurated and muddled. They

were granular kidneys, altered in appearance by a certain amount of chronic induration due to cardiac congestion. No gout in the great toe-joints."

CASE 10. Dilated and hypertrophied heart; mitral regurgitation; perihepatitis; ascites; high arterial pressure: urine normal; death.—Annie U—, æt. 49. Dr. Habershon (Clin. clerk, Mr. Thomas), December 28th—May 29th, 1879. Her father died from apoplexy. Her mother at an advanced age. One brother died from dropsy and heart disease. She does home work and has not drunk much. Never remembers being ill till five years ago, since then she has had cough and purulent expectoration. She has been feeling seriously weak and ill for the last year. About four months ago she noticed that her lower eyelids and cheeks began to swell, also her feet and ankles; the swelling of the face and eyelids was worse in the morning, that of her feet at night. She went under treatment but found her abdomen, thighs, and legs getting larger; the increased swelling caused her to apply for admission. On admission she is a fairly nourished woman. She sits propped up in bed, with a good deal of dyspnoea, face pale, and anxious, great œdema of legs and of trunk, especially in lumbar region; less œdema of arms; abdomen distended with ascites. Lungs resonant, but with prolonged expiration and sibilant rhonchi all over them, except at bases behind, where there are mucous râles. Heart's apex beats in fifth interspace, about half an inch external to nipple. Area of cardiac dulness increased. Loud systolic bruit at apex. Pulse 44, weak and irregular, very compressible. Very little urine passed; she sometimes remains twenty-four hours without passing any. It is rather light coloured, sp. gr. 1028, acid reaction, no albumen and no sugar. On January 3rd I made the following note:—"Pulse is long and persistent though small; the artery is not very full, but is discoverable during diastole. There is "safety-valve action of the mitral; occasionally there is a double heart beat to each pulse." January 15th.—She is still in the same general condition, but her pulse is 38, while her heart is 58 per minute. She has been taking digitalis four days; this is now stopped. January 28th.—Urine sp. gr. 1026, no albumen, large deposit of lithates. February 1st.—Paracen-

tesis to 10 pts. 6 oz. Much relieved. This was repeated on March 1st. Her condition remained obstinately the same; her urine, which was measured almost daily, could not be increased in quantity by any form of diuretic employed; the *Mist. Senegæ*, *Mist. Copaib. Res.*, and the diuretic pill, were all employed without avail; urine varied from 12 oz. to 18 oz. in the twenty-four hours. Paracentesis for the third time on April 15th. She has some severe bed sores. Paracentesis for the fourth time on May 29th. After this she sank rapidly and died.

This case closely resembles Cases 11 and 12 in my paper on the "*Clinical Aspects of Chronic Bright's Disease*," its characteristic features being bronchitis and heart failure, with capsulitis of the liver and obstinate ascites, all more or less directly the outcome of chronic Bright's disease.

Post mortem (by Dr. Hilton Fagge).—"Wasted upper part of body. Swollen lower limbs. A large quantity of fluid in right pleural cavity, compressing lower part of lung, which was covered with a honeycombed layer of vascular lymph. Lungs somewhat œdematous, otherwise healthy. A good deal of œdema in larynx, especially of left fold, also at base of tongue, in left side of soft palate, &c. Heart 15 oz., dilated rather than hypertrophied; left auricle decidedly hypertrophied and crisp; mitral valve healthy; aortic valves healthy; right auricular appendix filled with rounded softening thrombus. Peritoneum contained much straw-coloured fluid, and was generally thickened and slightly contracted; around the small intestine it was black. Liver 44 oz., perihepatitis, with an areolated separable layer; the tissue was rather cirrhotic. Spleen 8 oz. Kidneys 10 oz.; their tissue markedly blurred; thickened arteries; granular on the surface, but no indication of fatty epithelial changes. The microscope showed that there was extensive change, but I must confess that much of it appeared to be of a recent character, consisting of a cellular or nuclear infiltration. This was especially the case in the cortex, just beneath the capsule, where the greater part of the tissue was converted into a dense aggregation of nuclei. Also in deeper parts, around the Malpighian bodies, and around the vessels, and in some places without any obvious cause, the stroma was greatly swollen and infiltrated with nuclei. The tufts also were crowded with nuclei. A few of them were

degenerated and contracted. The stroma generally was, I think, decidedly thickened and more fibrous than usual, in some places very much so."

II. CASES OF LUNG FAILURE.

CASE 11. *Acute upon chronic bronchitis; hypertrophied and dilated heart; large granular kidneys, with acute epithelial changes, producing albuminuria thirteen days before death; urine previously normal; thick arteries; high arterial pressure; death.*—William W—, æt. 48. Dr. Habershon (Clin. clerk, M. Gilkes). April 18th—May 16th, 1879. His father died a young man, he believes from consumption; his mother had a joint affection which produced deformity of the joints affected; other family history unimportant. He was a carman for thirteen years, and after that a general labourer. He has been very subject to carbuncles, giving an account of seven of which he bears the scars. He says that he has been the subject of asthma, which in the winter of 1877 turned to bronchitis; he was in Stephen Ward under the care of Dr. Wilks. He then suffered from symptoms closely resembling those for which he is now admitted, and his urine was free from albumen. Has been a moderate drinker, and smokes about half an ounce of tobacco a day. He was laid up with the present illness five weeks after Christmas, since which time his cough has varied in severity, sometimes allowing him to work for a week or two. He is of average height. He is intensely cyanosed and has much dyspnoea. Sweats freely. Nails clubbed, fingers thick. Subconjunctival œdema. He coughs much and brings up frothy muco-purulent secretion. Hyper-resonance over front of chest, dulness at sides and back. Sibilant and sonorous rhonchus heard over front, mucous râles at back of chest. Heart's dulness increased, reaching half an inch beyond nipple, and for about four inches below, and also to the left of sternum; no bruits. "Pulse very persistent, long, and extremely high pressure." Urine sp. gr. 1030, dark coloured, loaded with lithates, no albumen. Temperature normal. April 22nd.—Urine sp. gr. 1030, loaded with lithates, no albumen. He still continues very livid;

respirations only 30; he is rather drowsy by day, restless at night. April 24th.—Expectorating bright blood, which on the next two days is rusty. April 26th.—I have a note as follows:—"Pulse still persistent but feels much *shorter*, he is not so well. His first sound appears to me like a presystolic bruit, such as I have heard occasionally in dilated hearts." April 28th.—Less blood in sputa; patient more cyanosed; feels better in the early morning, but has had another faint attack, which has lasted more or less all day. His pulse is very irregular and sometimes intermits. Expires very freely. Mucous râles all over back of chest, sibilant rhonchi in front. April 30th.—Urine small in quantity and loaded with lithates. May 3rd.—Patient feels weaker; his abdomen is filling out, is dull on percussion, pendulous, and fluctuation can be felt. Urine sp. gr. 1030, albumen $\frac{1}{4}$ th. May 6.—Respirations 32, temp. 96.8°. Urine sp. gr. 1034, has passed 18 oz. in last twenty-four hours, acid reaction, loaded with lithates, albumen $\frac{1}{4}$ th. Feels weaker; conjunctiva congested and œdematous. He steadily became worse; abdomen distended, legs œdematous, pulse too feeble to feel at wrist. Urine remained scanty and albuminous.

Post mortem (by Dr. Hilton Fagge).—"A large bulky man, with much dropsy of legs. Brain 56 oz., healthy, small arteries free from thickening. Lungs extremely emphysematous, especially the ear-shaped process of the left lung, which formed a large bladdery appendage connected with the lung by a narrow pedicle. There was a good deal of œdema. Bronchia velvety and full of pus, not generally dilated, although I found one tube in the right base which was so. Heart 20 oz.; muscle of left side soft and pale, of right firm, red, and indurated; a little atheroma on mitral valve, otherwise healthy; aortic valves healthy. Pulmonary artery as thick as aorta. Stomach intensely reddened (by mode of death). Liver congested in hepatic area. Spleen 8 oz., indurated. Kidneys 15 oz., deeply congested, but with very marked normal structure; their surface smooth and capsules thin. However, the microscope showed that they were far from being in a normal condition. I only found one Malpighian tuft degenerated, but the stroma was much increased, and in some parts the tubular structure seemed to be lost. In a pencilled preparation there was a

continuous plane of fibrillated material, dotted with fat granules and nuclei. The tubes were choked with granular matter. No doubt the epithelial changes were recent, coinciding with the congestive albuminuria, but I think there was older interstitial mischief."

CASE 12. Pleuritic effusion ; pulmonary apoplexy ; dilated and hypertrophied heart ; granular kidneys ; high arterial pressure ; urine normal ; death.—Helen E.—æ. 52. Dr. Pavy (Clin. clerk, Mr. Crosse). March 9th ; died March 23rd, 1880. An Irishwoman. Has lived in London since she was thirteen years of age. Married twice. She worked in white lead for ten years ; she ceased to work in it five years ago. She once had a blue line on her gums. Good health till twelve months ago, when she began to suffer from cough ; occasionally had hæmoptysis. Last November attended as an out-patient with very severe cough ; she spat blood at this time. On admission she is a thin, emaciated woman, looking ten years more than her age. She has great dyspnoea, and is expectorating dark, thick, tenacious, blood-stained, and frothy sputa. Heart's action frequent, sounds slapping. Pulse "frequent, small, long, and hard." General signs of bronchitis in the lungs.

March 12.—Urine sp. gr. 1020, no albumen, lithates. March 16th.—Urine sp. gr. 1015, no albumen, no lithates. Patient continues in much the same condition as on admission. Urgent dyspnoea. Great debility and exhaustion. No sleep. Takes but little food. On March 19th it is noted that the pulse is intermittent, dropping every fourth beat. Appears to be sinking. She is too ill to permit much examination. March 22nd.—Pulse very irregular, but much stronger. She wanders and is half unconscious. She died the following day.

Post-mortem examination (by myself).—"Emaciated. Slight œdema of legs. Right pleural cavity contained a layer of gelatinous material and serum, and was lined with corpuscular lymph. Left contained a considerable quantity of clear serum, but no signs of inflammation. Right lung : cicatricial nodule at apex, with slight emphysema over it ; at base the lung was so much compressed by fluid in pleura as to sink in water. Left lung : two ante-mortem thrombi were found in

branches of the pulmonary artery the size of a No. 4 catheter. One was apparently recent and there was no infarct in the lung. The other was of older date and more firmly adherent to the wall. There was a large infarct in the area of lung supplied by this vessel; this was situated at the base of the lung. The lungs were both œdematous, especially the left; both were indurated and shrunken looking. Bronchi and vessels thick and prominent. The tubes at the base of the right lung, in the collapsed portion, exuded pus freely. Heart $16\frac{1}{2}$ oz., enlarged, pale, and flabby looking; both ventricles equally enlarged; muscle pale, but not softened; left auricle considerably dilated; left ventricles dilated and thickened. The endocardium was thickened. The chordæ much thickened, greatly shortened, and matted together; the valve flaps were much thickened. The mitral orifice admitted only the tips of two fingers, it was distinctly stenosed. Aortic valves white and thick, otherwise healthy. Right ventricle and auricle a little dilated. Pulmonary artery white and thick. Aorta rather thick and inelastic, but no atheroma. Arteries generally very greatly thickened. Liver 44 oz., pale, friable, with a good deal of mottling from hepatic congestion, a rather atrophied-looking organ. Spleen 3 oz., small, firm, indurated, with thick arteries. Kidneys 5 oz., both of them typical specimens of true granular kidney; they were small highly granular organs, the granulation being perfectly even and regular throughout; the capsules somewhat adherent; the cortex very greatly wasted, reduced to one eighth of an inch. They were of an even red colour, there was no yellow mottling; no pallor of the cortex or hyperæmia of the pyramids. The arteries were very greatly thickened; the coats of the renal artery looked twice their normal thickness. Pelvis not dilated; ureters normal. In the cortex of one kidney was a small patch of atrophy following an old infarct, of which there were still some remains. No signs of gout in toe-joints. No external deformity of any joints."

CASE 13. *Bronchitis; œdema of lower extremities; jaundice; gall-stones; hypertrophied heart; granular kidneys; urine at first normal, afterwards a trace of albumen; death.*—William G—, æt. 43. Dr. Pavy (Clin. clerk, Mr. Bryden).

Admitted March 27th, 1878; died June 15th, 1878. Family history good. He is a clerk, has always been fairly healthy. He has been affected since boyhood with an angular curvature of the spine. Six weeks ago he was operated on for piles. No history of syphilis. He has drunk hard. Since his operation he has suffered from weakness, shortness of breath, and cough. He is a rather delicate, somewhat emaciated man, with a mild expression and slight tinge of redness on the cheeks, although he is generally anæmic. He presents the ordinary symptoms of bronchitis. Cardiac impulse jerky and diffused; the area of dulness may be slightly increased; the sounds appear muffled, the second is not clear. Radial pulse is somewhat frequent, hard, and regular. Urine light straw colour, sp. gr. 1015. No albumen or sugar, normal in quantity, phosphates are present. April 3rd.—There is dulness at left apex, deficient movement, and deficient entry of air. April 8th.—Legs began to swell yesterday evening, and are rather œdematous now. April 18th.—Urine sp. gr. 1015, some albumen present. April 23rd.—Lower extremities and scrotum much swollen. He has much difficulty in breathing. April 24th.—Jaundice first observed. April 26th.—Urine contains bile and a trace of albumen. May 6th.—The jaundice has passed away. The œdema of lower extremities, external genitals, and wall of abdomen persisted, his breathing became more difficult, and he died exhausted on June 15th.

Post mortem (by Dr. Fagge).—"The body was that of a heavy man with an angular curvature in the dorsal region. The lower limbs were very œdematous. The lungs contained much fluid. Heart 17 oz.; one of the aortic valves had a vegetation of considerable size upon it. Liver 61 oz., flattened in shape, with a transverse groove, probably caused by the distorted ribs. The ducts throughout the liver appeared slightly wider and more conspicuous on the cut surface than usual. I think the common duct was also widened, but the orifice into the duodenum was of natural size. In the common duct lay an angular black gall-stone; there were no others in the gall-bladder. Spleen 9 oz. Kidneys much wasted and very granular, one much smaller than the other."

CASE 14. *Bronchitis; hypertrophied heart; granular kidneys; high arterial pressure; urine normal; death.*—George T—, æt. 46. Dr. Wilks. November 22nd; died December 6th, 1879. Two months ago he complained of cold, pains in back, knees, &c. He had cough and dyspnœa, which caused him to apply to the hospital. On admission he had a congested face, a hard pulse, no dropsy, laboured respiration, coarse râles all over chest. Urine pale yellow, no albumen. Before death he was delirious, with very hard breathing, which stopped suddenly.

Post mortem (by Dr. Fagge).—"Body not wasted. Brain healthy. Chest contained three or four quarts of fluid. Lungs rather emphysematous. The larger tubes contained pus and the lung tissue was in places œdematous. Larynx healthy. Heart 25 oz.; left ventricle appeared not dilated. Liver 64 oz., nutmegged. Spleen 7 oz., very hard and fleshy. Stomach intensely congested. Kidneys 8 oz., very granular, wasted, red, with thick arteries. No gout in great toe-joints or knees."

CASE 15. *Bronchitis; dilated and hypertrophied heart; renal dropsy; urine usually normal, but albuminous on three occasions.*—Samuel D—, æt. 56. Dr. Pye-Smith (Clin. clerk, W. Spong). December 15th, 1879—March 24th, 1880. A labourer in Woolwich Arsenal. Family history good. He had rheumatic fever thirty years ago; temperate. He attended as an out-patient during January, 1879, with cough and expectoration. About three weeks before admission he noticed that he was short of breath on going upstairs; he left off work a week later (December 4th), when his legs had commenced to swell. On December 8th he noticed that his face was puffy and swollen especially under the lower eyelid; this lasted for three or four days, and then gradually subsided; meantime his legs continued to swell and his abdomen became enlarged. On admission he is described as anæmic, with blueness of lips and a distressed expression. He is sitting up in bed gasping for breath, but has no cough. There is marked subconjunctival œdema. Skin dry and harsh. There is more or less anasarca all over the body, especially in legs, thighs, and abdomen; penis and scrotum slightly œdematous.

There appears to be little or no ascites, though the abdomen is distended and tense, so that the umbilicus appears as a transverse slit. Liver enlarged, its lower edge can be felt about one inch above the umbilicus. Chest resonant except at bases behind, which are both dull. Expiration prolonged, breathing harsh, with râles at the bases. Cardiac dulness increased; apex beat one inch below and half an inch to outer side of nipple. (There appears to have been some doubt about this, as a marginal note says "Impulse feeble, not displaced;" on the other hand, it is later on again reported as much displaced). Heart sounds very indistinct and feeble, no bruit audible; sounds heard best at ensiform cartilage. Pulse small, weak, regular, and compressible. On December 24th.—I have myself noted, "Pulse small, but long and very incompressible, the harder you press the plainer it becomes." For four or five days before admission he is said to have passed very little urine. On admission it was dark coloured, loaded with lithates, sp. gr. 1015, slight trace of albumen. December 18th.—Urine sp. gr. 1012, clear amber coloured, contains no albumen, sugar or blood. *Microscopical examination.*—No casts can be seen, no blood-corpuscles, abundance of epithelium. By December 22nd much improvement had taken place. The œdema was much less, his urine having been steadily increasing in quantity. His leg, round the knee, measured 13 inches now, against 16 inches at time of admission. Heart sounds more distinct. Lungs as before; still dull at bases. December 29th.—Feet and legs only slightly œdematous. On January 13th he left the hospital for awhile, very much improved; the lungs had completely cleared up, though the respiratory murmur remained harsh. The urine had never been albuminous since the day after admission.

He was readmitted on February 24th. His condition is very much worse. His breathing is very difficult; he cannot lie down at night and he gets but little sleep; his cough is troublesome, with scanty, thick, bronchitic sputa. He has subconjunctival œdema, rather puffy lower eyelid, and dropsy of legs and feet. No œdema of scrotum or abdomen. Heart's impulse $2\frac{1}{2}$ inches below left nipple, and $1\frac{1}{2}$ external to it. Heart sounds muffled and indistinct action—"cantering;" first is very markedly reduplicated, and second is accentuated.

Pulse small and easily compressible. Lung sounds much as on previous occasion, but now there was thought to be slight dulness at right apex. Urine normal in quantity, amber coloured, acid, sp. gr. 1020, and contains no albumen. February 28th.—Yesterday afternoon patient had a very severe attack of dyspnoea, so bad that he appeared in danger for his life. The bowels had been very confined. He was dry cupped in twelve places, and an elaterium powder ordered, which purged his bowels thirteen times. This morning he is considerably better. His lung sounds are the same, the bases behind being deficient in resonance; rhonchus heard with inspiration and râles with expiration. Urine sp. gr. 1024, no albumen. Pulse feeble and regular, 86 per minute. March 4th.—Patient still complains of having to get out of bed at night because he cannot breathe. The following day œdema of the penis is noted; three days later this had disappeared. March 12th.—He is up and feels better. His condition steadily improved and he went out on March 24th. An account of his urine is appended; it was only albuminous on three occasions, once on first admission, and twice afterwards.

Date.	Quantity.	Sp. gr.	Solids.	Alb.
Dec. 16	... 20 oz.	... 1015	... 300	... Slight trace.
17	... 32 "	... 1012	... 384	... None.
18	... 46 "	... 1012	... 552	... "
19	... 60 "	... 1014	... 840	... "
20	... 60 "	... 1012	... 720	... "
21	... 72 "	... 1012	... 864	... "
22	... 70 "	... 1012	... 840	... "
23	... 70 "	... 1018	... 910	... "
24	... 50 "	... 1014	... 700	... "
25	... 52 "	... 1014	... 728	... "
26	... 60 "	... 1012	... 720	... "
27	... 72 "	... 1018	... 1080	... "
28	... 72 "	... 1015	... 1080	... "
29	... 64 "	... 1016	... 1024	... "
30	... 34 "	... 1015	... 510	... "
31	... 32 "	... 1016	... 512	... "
Jan. 1	... 32 "	... 1018	... 576	... "
2	... 28 "	... 1016	... 448	... "
3	... 30 "	... 1018	... 540	... "
4	... 22 "	... 1015	... 330	... "

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Date.	Quantity.	Sp. gr.	Solids.	Alb.
Jan. 5	... 26 oz. ...	1018	468	None.
6	... 30 " ...	1020	600	"
7	... 26 " ...	1020	520	"
8	... 22 " ...	1022	484	"
9	... 28 " ...	1020	560	"
10	... 32 " ...	1025	800	"
Pulv. Elaterii Sal., gr. xxx.				
11	Purged freely and much relieved.			
12	... 32 oz. ...	1025	800	"
13	... 32 " ...	1025	800	"
14	... 46 " ...	1025	1150	"
Went out.				
Feb. 25	... 46 " ...	1020	920	"
26	... 46 " ...	1021	966	"
27	... 40 " ...	1020	800	"
28	... 30 " ...	1024	720	"
29	... 28 " ...	1026	728	A trace.
Mar. 1	... 30 " ...	1022	660	"
2	... 36 " ...	1024	864	None.
3	... 35 " ...	1020	700	"
4	... 34 " ...	1024	816	"
Tinct. Digital., mx ; Mist. Senegæ, zj , ter die; followed by great improvement.				
5	... 40 oz. ...	1016	640	None.
6	... 28 " ...	1020	560	"
7	... 60 " ...	1015	900	"
8	... 60 " ...	1012	720	"
9	... 80 " ...	1014	1120	"
10	... 86 " ...	1018	1548	"
11	... 60 " ...	1014	840	"

CASE 16. *Chronic bronchitis; hypertrophied heart; high arterial pressure; urine normal.*—Catherine B—, æt. 38. Dr. Wilks (Clin. clerk, Mr. W. Spong). January 7th, 1880—February 26th, 1880. Has always had good health till four years ago, when she had rheumatic fever; never so well since, subject to occasional rheumatic pains and to coughs. For the last twelve months she has been more or less unwell; her heart has been irregular, sometimes "dropping a beat," and rendering her short of breath. For four weeks past she has had pain in lower part of her back over lumbar region. For the last two months she has lost flesh considerably.

She is a delicate, anæmic, pasty-looking woman. Lower eyelids puffy and slightly œdematous. There are well-marked

signs of bronchitis in her chest. Rhonchi heard all over and moist sounds abundant at the bases; resonance impaired at the bases behind. The heart's apex is much displaced, apex beat most distinct $1\frac{1}{2}$ inches below and 1 inch to outer side of nipple. No bruit. Second sound very much accentuated; best heard at base and towards the right side. Pulse long, regular, and incompressible.



Pressure $4\frac{1}{2}$ oz.

Her appetite is bad, and she frequently suffers from nausea and sometimes vomiting. Bowels usually constipated. She suffers much from headache, which appears to depend a good deal on her constipation. She is unable to hold her water more than two hours at a time, and is obliged to get up at night; she has noticed this since her last confinement. Her urine was perfectly normal during the time she remained in hospital. A chart of it is appended. Her cough improved, but never completely disappeared. She continued to be much troubled by headache. No cardiac bruit was ever discovered, but the second sound is always described as very much accentuated.

Date.		Quantity.		Sp. gr.		Solids.		Alb.
Jan. 9	...	56 oz.	...	1015	...	840	...	None.
10	...	40 "	...	1020	...	800	...	"
11	...	36 "	...	1020	...	720	...	"
12	...	36 "	...	1020	...	720	...	"
13	...	56 "	...	1015	...	840	...	"
14	...	40 "	...	1020	...	800	...	"
15	...	40 "	...	1016	...	649	...	"
16	...	—	...	1018	...	—	...	"
17	...	34 "	...	1020	...	680	...	"
18	...	32 "	...	1016	...	512	...	"
19	...	50 "	...	1018	...	900	...	"
20	...	28 "	...	1018	...	504	...	"
22	...	40 "	...	1018	...	720	...	"
23	...	30 "	...	1020	...	600	...	"
24	...	30 "	...	1022	...	660	...	"
25	...	30 "	...	1020	...	600	...	"
26	...	58 "	...	1020	...	1160	...	"

Date.	Quantity.	Sp. gr.	Solids.	Alb.
Jan. 27	... 44 oz. ...	1018 ...	792 ...	None.
28	... 44 „ ...	1018 ...	792 ...	„
29	... — ...	1018 ...	— ...	„
30	... 40 „ ...	1016 ...	640 ...	„
Feb. 1	... 30 „ ...	1016 ...	980 ...	„
2	... 18 „ ...	1020 ...	360 ...	„
3	... 20 „ ...	1020 ...	400 ...	„
4	... 16 „ ...	1026 ...	416 ...	„
5	... 20 „ ...	1022 ...	440 ...	„
6	... 34 „ ...	1020 ...	680 ...	„
14	... 20 „ ...	1020 ...	400 ...	„
18	... 26 „ ...	1028 ...	628 ...	„
20	... 24 „ ...	1025 ...	600 ...	„
21	... 44 „ ...	1015 ...	640 ...	„
22	... 22 „ ...	1016 ...	352 ...	„
23	... 30 „ ...	1020 ...	600 ...	„
24	... 18 „ ...	1020 ...	360 ...	„
25	... 26 „ ...	1018 ...	468 ...	„
26	... 25 „ ...	1016 ...	400 ...	„
27	... 21 „ ...	1015 ...	315 ...	„
28	... 25 „ ...	1015 ...	375 ...	„

CASE 17. *Chronic bronchitis and emphysema; hypertrophied heart; thickened and degenerate vessels; high arterial pressure; urine normal.*—Owen M—, æt. 70. Dr. Wilks (Clin. clerk, Mr. W. Spong). December 31st, 1879—January 23rd, 1880. Labourer in foreign grain warehouse. Has drank freely. Has been subject to cough for thirty years or more. For last two years it has been more severe and he has never lost it. For last twelve months has lost weight considerably. Has some difficulty of breathing. Sputum muco-purulent and abundant. Movements of chest impaired; respiration chiefly abdominal. Chest resonant all over. Rhonchi during both inspiration and expiration all over chest. Cardiac dulness diminished. Apex of heart much displaced, easily to be felt about 2 inches below and $1\frac{1}{2}$ inches to outer side of nipple. Heart regular, both sounds distinct, especially the second. No bruit to be heard. Pulse regular, hard, and incompressible; artery thickened and never quite empties itself, easily felt under the finger during diastole. Liver displaced downwards. He went out relieved of his bronchitis. His urine was never albuminous.

Date.	Quantity.	Sp. gr.	Solids.	Alb.
Jan. 8	... 40 oz. ...	1020 ...	800 ...	None.
9	... 50 „ ...	1020 ...	1000 ...	„
10	... 52 „ ...	1020 ...	1040 ...	„
11	... — ...	— ...	— ...	—
12	... — ...	— ...	— ...	—
13	... 40 „ ...	1012 ...	480 ...	None.
14	... 46 „ ...	1020 ...	920 ...	„
15	... 36 „ ...	1016 ...	576 ...	„
16	... 38 „ ...	1015 ...	570 ...	„
17	... 50 „ ...	1012 ...	600 ...	„
18	... 40 „ ...	1020 ...	800 ...	„
19	... 46 „ ...	1020 ...	920 ...	„
20	... 36 „ ...	1018 ...	648 ...	„
21	... 32 „ ...	1015 ...	480 ...	„
22	... 38 „ ...	1014 ...	532 ...	„

CASE 18. *Bronchitis; hypertrophied heart; high arterial pressure; urine normal.*—Thomas F—, æt. 66. Dr. Pye-Smith (Clin. clerk, Mr. Hind). December 31st, 1879—February 28th, 1880. Admitted for an exacerbation of chronic bronchitis, from which he has suffered for ten years. He has a good deal of inspiratory dyspnœa, with much muco-purulent expectoration. Rhonchi and moist râles in chest. Temperature normal. Heart's impulse difficult to feel, displaced about 2 inches below and $\frac{1}{2}$ inch to outer side of nipple. Pulse extremely long and persistent, occasionally intermittent. There was no œdema. His urine was normal in every respect during the whole time he was in the hospital. The urine chart appended is probably only approximately correct; the quantity of urine passed can only be taken as a minimum amount; a large amount was probably lost on several occasions.

Date.	Quantity.	Sp. gr.	Solids.	Alb.
Jan. 2	... 32 oz. ...	1015 ...	480 ...	None.
3	... 30 „ ...	1015 ...	450 ...	„
4	... 32 „ ...	1017 ...	544 ...	„
5	... 38 „ ...	1017 ...	646 ...	„
6	... 40 „ ...	1015 ...	600 ...	„
7	... 46 „ ...	1015 ...	690 ...	„
8	... 40 „ ...	1015 ...	600 ...	„
9	... 52 „ ...	1015 ...	780 ...	„
10	... 56 „ ...	1015 ...	840 ...	„
11	... 40 „ ...	1015 ...	600 ...	„

Date.	Quantity.	Sp. gr.	Solids.	Alb.
Jan. 12	... 46 oz.	... 1017	... 782	... None.
13	... 36 "	... 1020	... 720	... "
14	... 32 "	... 1015	... 480	... "
15	... 26 "	... 1015	... 390	... "
16	... 40 "	... 1015	... 600	... "
17	... 46 "	... 1020	... 920	... "
18	... 50 "	... 1017	... 850	... "
19	... 46 "	... 1020	... 920	... "
20	... 28 "	... 1020	... 560	... "
21	... 32 "	... 1020	... 640	... "
22	... 26 "	... 1015	... 390	... "
23	... 32 "	... 1015	... 480	... "
24	... —
25	... —
26	... 32 "	... 1020	... 640	... None.
27	... 26 "	... 1020	... 520	... "
28	... 32 "	... 1020	... 640	... "
29	... 40 "	... 1015	... 600	... "
30	... 50 "	... 1015	... 750	... "
31	... 46 "	... 1015	... 690	... "
Feb. 1	... 42 "	... 1017	... 714	... "
2	... 40 "	... 1017	... 680	... "
3	... 38 "	... 1017	... 646	... "
4	... 38 "	... 1015	... 570	... "
5	... 40 "	... 1015	... 600	... "
6	... 46 "	... 1015	... 690	... "
7	... —
8	... —
9	... 38 "	... 1015	... 570	... None.
10	... 40 "	... 1020	... 800	... "
11	... 52 "	... 1020	... 1040	... "
12	... 26 "	... 1020	... 540	... "
13	... 28 "	... 1020	... 560	... "
14	... 30 "	... 1020	... 600	... "
15	... 26 "	... 1020	... 520	... "
16	... 30 "	... 1020	... 600	... "
17	... 32 "	... 1020	... 640	... "
18	... 26 "	... 1020	... 520	... "
19	... 32 "	... 1020	... 640	... "
20	... 32 "	... 1020	... 640	... "

CASE 19.—*Bronchitis; emphysema; high arterial pressure; has previously had albuminuria; on present occasion urine normal.*—John H—, æt. 42. Dr. Pavy (Clin. clerk, Mr. A. Perkins). December 10th, 1879—January 10th, 1880. A labourer, His mother died, æt. 72, of chronic bronchitis.

He dates the bronchitis, for which he was admitted, from a severe illness in 1872, when he says he "took a cold," which laid him up for several weeks; he has been subject to bronchitis ever since. In January, 1879, he was in Clinical ward under Dr. Pavy, for bronchitis and emphysema. His urine was then loaded with albumen, high coloured, sp. gr. 1030. On the present occasion he is admitted with the ordinary symptoms of rather severe bronchitis. His aspect is highly characteristic of chronic Bright's disease. He has a somewhat pasty appearance, well-marked subconjunctival œdema, and a puffy lower eyelid, and flabby, pendulous cheeks. He has an emphysematous chest, and a good deal of bronchitis. Liver displaced downwards by lung. Heart's apex and dulness also obscured by lungs. Pulse very persistent, long, but easily compressed. It feels like a pulse of high pressure. His urine was never albuminous during his stay in hospital on this occasion, nor did it contain sugar; its quantity was excessive.

Date.	Quantity.	Sp. gr.	Solids.	Alb.
Dec. 29	... 66 oz. ...	1025 ...	1650 ...	None.
30	... 60 " ...	1025 ...	1500 ...	"
31	... 56 " ...	1020 ...	1120 ...	"
Jan. 1	... 70 " ...	1020 ...	1400 ...	"
2	... 76 " ...	1020 ...	1520 ...	"
3	... 68 " ...	1015 ...	1020 ...	"
4	... 74 " ...	1018 ...	1332 ...	"
5	... 70 " ...	1018 ...	1270 ...	"
6	... 82 " ...	1018 ...	1476 ...	"
7	... 80 " ...	1015 ...	1200 ...	"
8	... 100 " ...	1015 ...	1500 ...	"
9	... 90 " ...	1015 ...	1350 ...	"
10	... 86 " ...	1015 ...	1290 ...	"

CASE 20.—*Bronchitis; gangrene of lung and of toes from embolism; dilated heart, with ante-mortem thrombi; enteritis; peritonitis; kidneys, slight interstitial change; thick arteries; urine normal; death.*—Maurice O'C—, æt. 50. Dr. Pavy (Clin. clerk, Mr. Anderson). March 20th, 1879; died April 1st, 1879. A carman. Family history unknown. He had erysipelas two and a half years ago, following a kick from a horse. A year ago he was ill for four days, when he had great difficulty of breathing and a cough; otherwise health

has been good. His present illness commenced suddenly, two months ago, with a cold sweat and feeling of great weakness; no shivering. He had a good deal of cough, the sputa being frothy and white. For last fortnight he has been worse, the sputa being blood stained; he has felt very weak and ill lately, and has had no rest at night, on account of his cough. He is a pale, thin-looking man; he is compelled to sit up in bed, breathes rapidly and with difficulty, expectorating a quantity of blood-stained mucus streaked here and there with purulent material. His severe illness prevents a very complete examination of his chest, but it appears to be resonant all over; the breathing is described by the clerk as "bronchial" all over the chest, probably he means "bronchitic;" in other words, coarse, and with slight rhonchus; moist râles are also heard, more especially at extreme right base. The heart appears to be dilated, action quick and tumultuous; six or seven beats rapidly succeed each other, followed by a pause, and then more rapid beats. Something very like a presystolic bruit is heard at apex, and the second sound is rough, prolonged, and accentuated; the heart sounds are much obscured by the breathing. I make a note that "he has a persistent pulse with thick vessels; there is a sound very like a presystolic murmur, but I do not think *quite* like it. I fancy it is a reduplicated first sound. Chronic Bright's disease, with dilated heart from high pressure." His urine contains a quantity of lithates, sp. gr. 1030, no albumen. Mental faculties clear. Temperature 99.2°. March 21st.—"Dr. Pavy saw patient to-day, and detected a short presystolic bruit and fine moist râles at base of right lung." Heart still very tumultuous, and breathing difficult. He was treated chiefly by digitalis, and appeared to improve daily until April 1st, when he was found to be much worse, pulse very irregular, and breathing hurried. Death occurred that evening. Unfortunately no further note occurs in the report about the urine after the first day.

Post mortem (by Dr. Hilton Fagge).—"All the right toes are gangrenous and horribly fetid. Lungs bulky and very œdematous, but they were also affected by extensive, scattered, ill-defined gangrene. The most definite patch was at the right apex, where there was a considerable cavity, with a slough hanging suspended from a single point. But in most

places the gangrenous parts were continuous with those which were not so, and were indicated chiefly by their colour and the dirty green brown fluid which freely exuded, unlike the clear serum which oozed from the rest of the lung. (It appeared to be just the sort of affection which would arise from sucking in of vomited matters, but I could not learn that vomiting had been present). Heart 17 oz., dilated rather than hypertrophied, its tissue soft and rather yellow. In the apex of the ventricle were several white ante-mortem thrombi of various sizes entangled in the muscoli papillares or free in the cavity. Valves healthy. The popliteal artery was dissected out, but hardly low enough; near its bifurcation it contained a firm piece of clot, which I thought to be the upper end of an embolus. There was general acute peritonitis, the intestines being matted together with recent lymph. The lower part of the small intestine was firmly wedged into the pelvis; the greater part of it was of an intense purple colour, and covered with lymph. After removal, the whole thickness of the gut was found to be in a most intensely inflamed condition. The mucous membrane was of an ashy-grey colour, rough, and dry looking. There were two patches of this affection, each occupying a foot or two of the length of the bowel; the lower one included the cæcum, the upper one was three or four feet higher up. Evidently this enteritis ("diphtheritic" in character) was the cause of the acute peritonitis. The appendix cæci contained pus, but was not swollen or reddened. Liver 6½ oz., fatty, and also rather granular on section, probably a little cirrhotic. Spleen, 5 oz., firm, contained an infarct. Kidneys, 13 oz., contained several infarcts; they were rather flabby organs, but showed a good structure in their cortex; *the arteries were rather thick and quill-like*. A microscopical section made by Dr. Mahomed was thought to show some intertubular thickening, but it seemed to me that this was very doubtful."

My notes about the kidney are as follows:

"Chronic tubal nephritis (?), tubes full of epithelium. Capsules but little thickened. Arteries much thickened, but little intertubular change." A microscopic section accompanies this paper. I may here remark that all my notes as given here are made without any knowledge of the kidney or case

from which the section has been taken. I have been careful to work my material by numbers, and so mix the cases over long periods that I can only identify them after reference by means of the number. The observations are therefore entirely unprejudiced. On a second examination I find that I have not done justice to the really large amount of intertubular change present in this section.

CASE 21. *Phthisis ; gout ; general atheroma ; very granular kidneys ; heart only 11 oz. ; urine normal ; death.* Robert C—, æt. 65. Dr. Goodhart (Clin. clerk, Mr. A. P. Hills). March 5th ; died April 4th, 1879. A warehouseman, but has never done any heavy work. His father died of chronic bronchitis at seventy-nine, his mother at sixty-nine, cause unknown. He has lost three brothers, of whom two are said to have died of "decline," aged seventy-two and thirty-four respectively. He is a temperate man. In 1876 he was in Guy's under the care of Dr. Pavy with rather severe bronchitis, which affected the right lung most ; on this side there was some consolidation ; bronchial breathing and bronchophony were heard at right apex, and some dulness and crepitations at right base behind ; elsewhere in chest the ordinary signs of bronchitis. His urine then was 1022 sp. gr. and free from albumen. He continued well after this till the middle of 1878, when he again fell ill with cough ; getting weaker, he determined to apply for admission. He lies mostly on his back, looks depressed, weak and ill. Dilated vessels on cheeks. No œdema. Some signs of phthisis at both apices, especially left. Cardiac impulse scarcely perceptible ; dulness normal ; sounds normal, rather feeble. Pulse 84, radial arteries very irregular, hard, and beaded.

Pressure 3 oz.

He had two attacks of paralysis ; the first, six years ago, was confined to right arm, the second was last summer, affecting the same limb and lasting only a very short time ; it had passed off entirely in two or three days. Urine 1016, no

albumen, sugar, or blood. March 20th and 26th.—No albumen in urine. He died on April 4th.

Post mortem (by Dr. Hilton Fagge).—"Body spare. Brain healthy. Recent pleurisy on left side. Lungs affected with a markedly tubercular form of phthisis; scarcely any tendency to caseate. The tubercles in clusters, chiefly in the upper lobes. Larynx healthy. Bronchial glands enormously swollen, blackish grey, firm, and hard. Liver 72 oz., very fatty. Heart 11 oz., not hypertrophied; aorta affected with a moderate degree of arteritis deformans. Kidneys 9 oz., very granular, with many cysts, and a very wasted cortex. Gout in toes. Intestine presented numerous tubercular ulcers with much abundant subserous tubercle."

III.—CASES OF CEREBRAL DISEASE.

CASE 22. Cerebral hæmorrhage; general arterial disease of peripheral vessels; hypertrophied and dilated heart; kidneys healthy; urine normal; death.—James G—, æt. 55. Dr. Pavy. Admitted October 8th, 1879; died October 9th, 1879. He was admitted in an early comatose state, and speedily lapsed into a state of complete paralysis. No complete report was made of his case.

Post mortem (by Dr. Goodhart).—"Body exceedingly fat. Cranial bones very thick and heavy; the dura mater firmly adherent to the skull; arteries of brain very bad; the internal carotid on each side very large and standing open rigidly. All the vessels were in a similar state, many atheromatous and calcareous plates being studded through them. Brain 48 oz. A very large smash into left hemisphere; an ounce of blood clot was removed. This had torn up the corpus striatum, and escaped into the ventricle; in addition there was extensive ecchymosis and hæmorrhage into the pons. Pleuræ healthy. Lungs œdematous, a little pus in the smaller tubes. Intense ecchymosis under the tracheal mucous membrane throughout its whole length. Heart 22 oz., very dilated and hypertrophied left ventricle; both mitral and aortic valves markedly thickened at their edges; aorta very fair. Arteries of both upper and lower limbs atheromatous,

Vessels of tongue on transverse section very thick indeed. Liver 10½ oz., fatty, vessels thick. Spleen 10½ oz., normal. Kidneys 15½ oz., surfaces smooth. They contained a few minute cysts, but looked very good organs indeed. They were very congested, probably from the mode of death. No urate of soda in joints of great toe."

CASE 23. *Cerebral hæmorrhage; general arterial disease; heart slightly hypertrophied; kidneys healthy; vessels thick; urine normal; death.*—G. Q—, æt. 55. Dr. Wilks (Clin. clerk, Mr. Marsh). November 18th; died November 24th, 1880. A carpenter. Has always been steady and sober. Has never had rheumatism. Fatty tumour removed from right shoulder nine years ago. From about this time he has been subject to tremor in his right hand and forearm; this trembling has increased lately. For the last two or three weeks he has had numbness in left leg and foot, especially over heel. No fainting fits or other signs of ill health. Was taken suddenly with a fit on the day of admission. (The details of which I need not relate.) The heart's impulse widely diffused over the left half of the chest. There is a diastolic bruit in aortic area, heard also in large vessels of neck. The pulse is regular, 80, and full. (I did not see this case, and therefore have no exact note about the arterial pressure.) He had Cheyne-Stokes' respiration. On the day after the fit it was found that he had paralysis of left arm, leg, and face, and rigidity and tremor of the right. Contraction of left pupil. His urine could not be saved on the first two days as he passed it into the bed. November 20th.—The urine was drawn off and found of slightly acid reaction, sp. gr. 1014, no albumen. On the 22nd it had exactly the same characters. On November 23rd his urine was found to be bloody, probably the results of his catheterisation, and it was still more so on the 24th. His respirations still continued to exhibit the Cheyne-Stokes' phenomena. He died on this day.

Post mortem (by Dr. Goodhart).—"Spare, grey hair, bald. No dropsy. The vessels at the base of the brain had a thick opaque appearance, as if sodden in water. There was one little patch of atheroma at the bifurcation of the basilar, but not elsewhere. They were really conspicuous for an absence of

atheroma, though generally thick. The right hemisphere was filled out and the convolutions flattened; the membranes over the convolutions thick on both sides. There was no meningeal apoplexy. A section of the brain showed a large extravasation of blood into the right temporo-sphenoidal lobe, converting it into a cyst. Upon making horizontal sections of the large ganglia and brain it was apparent that the hæmorrhage had come from a spot in the internal capsule and hinder part of the corpus striatum, and had just involved a small portion of the optic thalamus. All the blood appeared to be of one date and recent; the adjacent brain was stained yellow from soakage. The vessels were examined in various parts of the brain for miliary aneurisms, but none were found. Numerous punctiform hæmorrhages on under surface of fornix. Lungs: extreme congestion of both, and early broncho-pneumonia. Heart, 12½ oz; left ventricle perhaps a little thick for its size, and in the muscular wall were seen small patches of atheroma; the valves were all competent. Aorta and vessels: extreme soft leathery thickening of the whole aorta; the inner surface being rugose and grey in colour; the coats as a whole much thickened. The artery was dilated, its circumference being four and a half inches. About an inch above the aortic valves, on the convexity of this arch, was a vertically elongated aneurismal dilatation (about the size of a pigeon's egg). There was another aneurism at the innominate orifice, of an inch in diameter, the great vessels of the neck being very thick. The whole aorta, descending as well as ascending, was very atheromatous, and the same may be said of the femoral and iliac arteries. The radials were also thick and soft, and patched with yellow. Stomach and intestines healthy. Liver, 56 oz., healthy. Gall-bladder full. Mesenteric and lumbar glands healthy. Spleen 5 oz., some thickening of its capsule. Supra-renal capsules healthy. Kidneys 12 oz., vessels thick and decidedly atheromatous; the cortex was smooth and looked quite healthy, but there were one or two cysts of small size visible. No urate of soda in any of the joints.

The vessels and kidney were reserved for microscopical examination, but were unfortunately thrown away by mistake."

CASE 24.—*Right hemiplegia from cerebral hæmorrhage ; hypertrophied heart ; high arterial pressure ; urine normal.*—Henry G—, æt. 61. Dr. Wilks. (Clin. clerks, Mr. Warner and Mr. A. G. Mahomed). May 11th, 1880—September 2nd, 1810. A sawyer. Has generally had very good health. Two days before admission, on getting up in the morning, he found that he had lost power greatly in the right leg and arm ; by the evening he had lost power so much that he was unable to stand. On admission he cannot use his right arm and leg at all ; the leg lies in a helpless condition rotated outwards, the arm pronated. There is very little facial paralysis. Sensation is perfect everywhere. Speech is rather thick and indistinct, but there is no aphasia. There is well marked subconjunctival œdema ; his lower lid is slightly puffy. Pulse long, full, and persistent ; requires considerable pressure to obliterate it ; the vessel is not particularly thickened.



Pressure 4 to 6 oz.

Heart's apex very indistinct, one and a half inches directly below nipple. At apex the second sound is accentuated, and a faint systolic bruit can be heard (only in expiration). The area of cardiac dulness is encroached upon by emphysematous lung ; there is deficient expansion and considerable in-drawing of the chest walls during inspiration. Breath sounds are deficient over the margins of the lungs in front. He passes his urine more frequently at night than he used to do ; it is normal in all respects. He slowly but steadily regained strength in his right arm and leg, and when he left hospital about three and a half months after his attack, he was able to use them very fairly well. His pulse tracing taken during August shows that his arterial pressure was unduly high ; while his urine chart indicates very frequent variations in the quality of his urine, its specific gravity falling at frequent intervals to 1010 or 1012. No albumen was ever discovered in it, but it usually gave a cloud with tannic acid and perchloride of

mercury, and it was observed that this cloud was most dense when the specific gravity was lowest. The urine frequently deposited uric acid crystals.

Date.	Quantity.	Sp. gr.	Solids.	Alb.	Bowels.
May 13	40 oz.	1025	1000	None	...
14	40 "	1027	1080	"	...
15	40 "	1026	1040	"	...
16	44 "	1025	1080	"	...
17	40 "	1027	1080	"	...
18	36 "	1027	972	"	...
19	30 "	1026	780	"	...
20	36 "	1027	972	"	...
24	30 "	1025	750	"	...
25	32 "	—	—	—	...
26	18 "	—	—	—	...

Diarrhoea.

June 3	24 oz.	1027	648	None	1
4	40 "	1023	920	"	0
5	54 "	—	—	—	...
6	28 "	—	—	—	1
7	30 "	—	—	—	...
8	40 "	1025	1000	None	1
9	44 "	1025	1100	"	1
10	50 "	1027	1350	"	1
11	50 "	1015	750	"	1
July 1	44 "	1012	528	"	1
2	46 "	1020	920	"	2
3	64 "	1020	1280	"	1
4	60 "	1015	900	"	1
5	54 "	1018	972	"	1
6	58 "	1015	870	"	1
7	50 "	1015	750	"	1
8	46 "	1012	552	"	1
9	46 "	—	—	—	...
10	58 "	1020	1160	"	1
11	70 "	—	—	"	0
12	58 "	1015	870	"	2
13	40 "	1015	600	"	1
15	72 "	1015	430	"	1
16	24 "	1020	480	"	1
25	50 "	—	—	—	...
26	52 "	—	—	—	...
27	76 "	1015	1140	"	1
28	—	1012	—	"	...
29	56 "	1015	840	"	1
30	68 "	1012	816	"	1

Date.	Quantity.	Sp. gr.	Solids.	Alb.	Bowels.
July 31	... 70 oz.	... 1009	... 840	... None.	... 1
Aug. 1	... 56 "	... —	... —	... —	... —
2	... 60 "	... 1016	... 960	... "	... 1
3	... 68 "	... 1010	... 680	... "	... 1
4	... 64 "	... 1012	... 768	... "	... 1
5	... 60 "	... 1012	... 720	... "	... 1
6	... 66 "	... —	... —	... —	... —
7	... 32 "	... 1024	... 768	... "	... 1
8	... 64 "	... —	... —	... —	... —
9	... 50 "	... 1022	... 1100	... "	... 1
10	... 56 "	... 1016	... 896	... "	... —
11	... —	... 1020	... —	... "	... 1
12	... 40 "	... 1020	... 800	... "	... 1
13	... 58 "	... 1020	... 1160	... "	... 1
14	... 50 "	... 1012	... 600	... "	... 1
15	... 60 "	... 1020	... 1200	... "	... 1
16	... 74 "	... 1012	... 888	... "	... 1

CASE 25. *Right hemiplegia with partial aphasia from cerebral hæmorrhage; thick and atheromatous vessels; high arterial pressure; urine albuminous for three days after attack, afterwards normal.*—William O—, æt. 59. Dr. Wilks (Clin. clerk, Mr. Prentice). Admitted March 9th, 1880; discharged April 19th, 1880. His father is said to have died from rheumatic gout. He has had no severe illnesses. No syphilis or gout. Sixteen days before admission he was sweeping a road, when he suddenly felt as if he were drunk, had to stand against a house to prevent his falling. When he came to himself he found that he had lost the use of his right arm, and partially of his right leg; his speech also was thick. On admission he is a tall man, with a very red face. Subconjunctival œdema. Arcus senilis. Partial paralysis of right face, arm, and leg, with some aphasia. Chest hyper-resonant, cardiac dulness obliterated. Heart sounds distant. Heart's impulse scarcely perceptible. Pulse remarkably long, artery tortuous and thickened. Urine normal colour, acid, sp. gr. 1015, albuminous, no sugar. March 12th.—Urine sp. gr. 1016, still albuminous. After this date albumen was never found in the urine; it was examined and noted on March 13th, 15th, 17th, 18th, 19th, 23rd, 25th (sp. gr. 1015), 31st (sp. gr. 1020), April 6th, 8th, and 17th.

CASE 26. *Right hemiplegia and aphasia from cerebral hæmorrhage; thick and atheromatous arteries; high arterial pressure; urine normal.*—William S—, æt. 50. Dr. Habershon (Clin. clerk, Mr. A. Scott). October 28th; died December 9th, 1879. His father died in an apoplectic fit. One brother had an attack similar to patient's, was paralysed five years, and then died in another fit. Mother died of old age. He is a coffee-stall keeper. Has drank very hard; was drunk two days before present attack. He was suddenly attacked at 5 a.m. on the morning of admission. He jumped out of bed and made some inarticulate moans. His wife got up, and found that his right leg was drawn up, and the right side of his face was "working;" he was standing on his left leg and supporting himself by a chair. His wife helped him into the chair, and he indistinctly uttered the words "fit" and "hospital." After that he gradually lapsed into a state of stupor but occasionally made signs for drink, and the chamber-vessel which he used. I need not detail his symptoms; let it suffice to say that he had right hemiplegia and aphasia, that it was possible to rouse him, but he otherwise appeared in a state of stupor. His pulse was 76, hard, and resisting. Brachial arteries hard and cord-like. Heart's impulse can only be seen and felt in epigastrium; area of dullness normal, sounds almost completely obscured by laryngeal sounds, but seem clear. Urine acid, no albumen. November 2nd.—Urine, sp. gr. 1018, acid, no albumen. Granular casts. It was difficult to get any urine as he passed everything under him. He was removed to the infirmary, able to answer questions by gestures, but not to speak; the right arm and leg were flexed and rigid.

CASE 27. *Right hemiplegia from cerebral hæmorrhage; hypertrophied heart; high arterial pressure; urine normal.*—James G—, æt. 68. Dr. Pye-Smith (Clin. clerk, Mr. Sturge). June 21st—July 2nd, 1879. Father died of dropsy, æt. 50. Mother died of mortification of leg after a fall. Has had good health till the last two winters, during which he has suffered from cough and shortness of breath. He was suddenly taken this morning with paralysis of the right arm and face and partial paralysis of the right leg. He

never lost consciousness. Area of cardiac dulness increased, including left nipple. Impulse felt plainly in nipple line below fifth rib, and a shock, quite perceptible below sixth rib in same line. First sound thick, second normal. Pulse 60, full, throbbing, easily compressed; but when the vessel is emptied it can be felt like a cord and rolled under the finger.



Pressure $2\frac{1}{2}$ oz.

Urine straw coloured, sp. gr. 1012, no albumen. June 24th. —Urine loaded with lithates. He went out, able to walk fairly well, but with considerable weakness of right arm; he had recovered his speech.

CASE 28. *Cerebral softening; extremely high arterial pressure; urine occasionally albuminous.*—John M—, æt. 52. Dr. Habershon (Clin. clerk, Mr. Combe). April 22nd—May 19th, 1879. His father died at an advanced age of disease of the prostate, his mother from chronic bronchitis. One brother and two sisters thought to be consumptive; all his family are said to be very nervous, but none of them subject to any kinds of fits or rheumatism. Lately he has kept a coffee house, formerly a beer-shop. He says he has been abstemious, but not much reliance can be placed on him. Ever since the age of seventeen has suffered from nasal polypi, and has had twenty removed, the last about fourteen months ago. About five years ago he noticed that his speech was not so fluent as formerly; he seemed to have lost some control over his tongue. Seven or eight months ago he found he could not sleep at night, and for three months took laudanum, taking at last, he says, as much as $\frac{1}{2}$ oz. at a time. During the last two and a half months he has taken chloral instead. Six or seven weeks ago he was noticed to walk imperfectly with his left leg, which symptom has gradually increased. A well-made man, above the average height (5 ft.

10½ in.) ; he appears well nourished, but he says he has lost flesh lately. Cardiac dulness normal. Apex beat one inch below and to the inner side of nipple. Heart's action regular. Pulse 92, and very persistent. The condition of this man's pulse was very striking ; the pulse was long, very incompressible, and extremely persistent ; the vessel appeared to be thickened. Bowels confined for a week. He is extremely forgetful and stupid ; he appears to be in low spirits, and has typically the aspect and manner characteristic of softening of the brain. Movements of left leg impaired. He is sleepless at nights and very nervous. Urine 1018, clear, good colour, it contains a trace of albumen. April 26th.—Sp. gr. 1018, albumen slightly increased. May 1st.—Urine, sp. gr. 1020, albumen a trace. April 7th.—Speech much better. He is gaining power in left leg. Urine pale and clear, sp. gr. 1021, not a trace of albumen. April 13th.—Urine normal. April 19th.—Contains a slight trace of albumen. He went out, his general condition remaining unchanged. July 5th.—He was readmitted with just the same symptoms as before ; unfortunately, there is no note of his urine on this occasion. He went out on August 3rd, 1879. In this case the very slight trace of albumen present was overlooked till I examined it myself, being led to do so by the very extreme arterial pressure.

CASE 29. *Cerebral softening ; old left hemiplegia ; very high arterial pressure ; hypertrophied heart ; low specific gravity ; no albumen.*—Robert H—, æt. 64. Dr. Moxon (Clin. clerk, Mr. L. Lane). July 2nd—September 14th, 1879. A clothes dealer. For the last seven months his memory has been failing him ; he has been in the habit of doing foolish things, has been absent-minded and readily excited to laugh at trifles. He has complained much of headache. About four months ago he had sudden hemiplegia and hemianæsthesia without loss of consciousness. In about two months he regained sensation and partially motion. He is a tall, skinny, bleary-eyed man, with white hair, looking rather more than his age. His temporal arteries are prominent and tortuous ; his pulse 64 per minute, is strikingly persistent, tortuous, incompressible and long ; the arterial coats are distinctly thickened. Apex

beat half an inch below and in nipple line. Second sound sharp at base. There is partial left hemiplegia; sensation is normal on the affected side. He complains much of headache in right temporal region, with some vertigo and considerable loss of memory. Urine, sp. gr. 1010; no albumen. July 14th.—Sp. gr. 1010, no albumen. July 15th.—Sp. gr. 1011, no albumen. July 21st.—Sp. gr. 1011, no albumen. July 28th.—Sp. gr. 1010, no albumen.

He suffered very severe pain in right temple and right eye, the scalp being tender on percussion. He was leeches several times for this with some relief; but he received the most permanent benefit from a course of purging with mercurial, rhubarb, and podophyllin pill, and a mixture containing sulphate of iron and magnesia. No albumen was ever detected in the urine, but the specific gravity was uniformly low. He was so forgetful and stupid that he could not be made to save it.

CASE 30. *Attack of unconsciousness; headache; vertigo; hypertrophied heart; high arterial pressure; urine normal.*—Eliza S—, æt. 64. Dr. Pye-Smith (Clin. clerk, Mr. Currah). December 26th, 1879—March 26th, 1880. She has had good health all her life. Lately she has been subject to attacks of vertigo, and has sometimes been obliged to sit down to prevent herself falling. On the day of admission was seized, while in the street, with an attack of vertigo, and was obliged to support herself against some railings; soon after she fell down and lost all consciousness. She does not recollect anything more till she found herself in bed in the ward; she was two hours in the hospital before she recovered consciousness. For the last two days she has suffered from severe shooting and throbbing pain in her head; this pain is increased by movement and pressure. She has not lost sensation or power of movement in any of her limbs. Heart's impulse slow and heaving, most marked in nipple line, in fifth interspace; first sound feeble, almost absent, second extremely loud over upper part of sternum; no bruit. Arteries tortuous and much thickened; strong pulsation in episternal notch and in right carotid and subclavian. Pulse 70, extremely persistent, long, and rather incompressible.



Pressure 4 oz., takes more.

Urine, sp. gr. 1025, it contains no albumen. Temperature 97·8°. While in the hospital she suffered much from headache, deafness in the right ear, and noises in the head. It was some days before she could get up and about the ward. She had a rather severe attack of diarrhœa towards the end of January, which seems to have done her good. The condition of her urine is but seldom referred to, but on four occasions it is noted as free from albumen, and the specific gravity is given as 1025, 1015, 1012, 1014, the last three observations being made in March shortly before she went out.

IV. CASES OF RENAL DROPSY, WITHOUT ALBUMINURIA.

CASE 31. *Chronic renal œdema (simulating myxœdema); high arterial pressure; general fibrosis; thickened arteries; hypertrophied heart; granular kidneys; urine normal; death.*—William F—, æt. 42. Dr. Wilks (Clin. clerk, Mr. Starling). November 19th—Died December 31st, 1879. A lighterman till three years ago, when he became a night watchman on the Swan Pier. Father asthmatical, died at 80. Mother is now 70, spits blood, has dropsy in the legs. Two brothers have died of diabetes. He never drinks beer or spirits. He was a strong and healthy man till nine years ago, when he had erysipelas, his face then remained swollen for ten weeks. When first able to get up and go out after this he was seized with pain in his left leg, which laid him up for another three weeks. He had gout in his great toe-joints two years ago. He had several attacks of giddiness, in one of which he fell overboard into the river; on account of this he gave up his work as lighterman. For the last four or five months his memory has been failing him. He cannot hold his water for long; as soon as the call to pass it comes, he must micturate or it dribbles from him.

Patient is a well-developed, well-nourished man, com-

plexion florid, hair spare, fine and straight, tending to baldness. Eyelids very puffy, face, hands and feet swollen. Much capillary congestion of cheeks. Skin thick. The swelling is not like that of ordinary œdema, it is more resistant and elastic. No topi in ears. Teeth worn down. No chalk stones or grating of any joint, though the left great toe-joint is painful on pressure. Lungs normal. Apex of heart cannot be felt; area of dulness is not increased. First sound normal, second reduplicated. Pulse 66, regular, long, and persistent; pulse tracing shows extremely high arterial tension.



Pressure 5 oz.

Urine light colour, sp. gr. 1020, no albumen or sugar, acid reaction, no casts. Sight good. Hearing a little defective. Voice thick and speech deliberate. Memory bad.

December 10th.—Complains of more giddiness, pain in right shoulder and left hip like rheumatism. December 14th.—Sleeps a good deal and does not take his food well, otherwise in same state. The eyelids of both eyes are now as swollen as when he was admitted, and hang down when the eyes are closed, so that the upper ones droop nearly on to the lower lids. It has hardly the feel of fluid, more like mucus, though it gravitates to the side on which he is lying. December 16th.—Coincidentally with the appearance of bile in the urine and slight jaundice albumen also appeared, but for one day only. December 19th.—Nearly all the œdema of eyelids has disappeared. There is a slight blush on right cheek and above eyebrow. Temperature normal. Hands and skin generally very pale. December 20th.—Cheeks, nose, and region about eyes have a diffused red blush, somewhat like erysipelas; the nose is also swollen. Undefined sense of general pain still present. December 30th.—Reddish blush on left hip; skin not warm, but feels dry, and has a wrinkled aspect. Needs to have any question repeated and shouted to him before he answers, and then he speaks in a slow drawling manner. December 31st.—Last night he rolled about the bed almost

throwing himself off; is very cold; swelling of eyelids reappeared, and they feel as if full of jelly. Is very drowsy and sleepy, cannot be roused to answer any questions, even when shouted in his ear; and when told to put out his tongue makes a drawling sort of noise, sounding like "What, sir." Pulse feeble. He died this day.

Date.		Quantity.		Sp. gr.		Solids.		Alb.
Nov. 20	...	38 oz.	...	1020	...	760	...	None.
21	...	20 "	...	—	...	—	...	"
22	...	88 "	
23	...	86 "	
24	...	56 "	
25	...	48 "	...	1017	...	816	...	"
29	...	54 "	
30	...	44 "	
Dec. 2	...	44 "	
3	...	48 "	
14	...	—	...	—	...	—	...	"
15	...	—	...	—	...	—	...	None.
16	...	26 "	...	—	...	—	...	Albumen and bile.
18	...	44 "	...	—	...	—	...	None.

Post mortem (by Dr. Goodhart).—"A big man, with puffy appearance, and very œdematous state of eyelids. All the body looked puffy, but there was no dropsy, no pitting on pressure. In some parts, but by no means in many, or even in most, there was a fluid jelly material between the muscles, some of which I collected. It was mostly in the inter-muscular septa of the thighs, and about the pectorals. In the subcutaneous fat there was nothing abnormal, no excess, no œdema of any kind. The bones of the cranium were a little thick and a little rough perhaps. Membranes of brain perfectly healthy, and except that one part of the left vertebral was a little atheromatous, there was no disease whatever; they were all thin, good vessels. The brain weighed 51 oz. and looked remarkably good, but it cut with a peculiar toughness, somewhat like cartilage in its resistance, and some parts were more so than others. I noticed particularly that the right cerebellum was so, yet this occurred without any appreciable alteration to the naked eye. The brain looked perfectly healthy. I should say, that I had pointed this out before I had any idea of the supposed nature of the case, or, indeed,

any preconception in any direction of what I should find in the body. The retina in each eye was perfectly healthy. Cervical glands, thymus, and thyroid normal. Costal cartilages healthy. Each pleura contained a large quantity of serous fluid, no lymph. Both lungs much compressed by fluid in the pleura; the left lower lobe was quite airless, and the right nearly so. With this exception the lung tissue was healthy. A very little œdema of the ary-epiglottic folds, nothing sufficient to cause obstruction. Mediastinum somewhat gelatinous. Pericardium contained a good deal of serum, the serous membrane was thick and sodden-looking from the long-continuing presence of fluid in it. The heart weighed 15 oz. Its cavities, particularly the left ventricle, were thick and dilated; there was free regurgitation through the mitral as tested by water, though the orifice was actually of normal size. The muscular tissue was of a peculiar appearance, being brown in colour and translucent or gelatinous. It looked juicy and streaked with a few fibrous bands. The valves were quite healthy and the aorta also. The stomach and all the intestines were thickened from an œdematous condition, just such a state as is often seen in old-standing ascites or renal anasarca, but in this case there was no noticeable ascites. Liver 92 oz., dark, reddish-brown, firm; gelatinous translucency similar to that of the heart. No lardaceous disease. Pancreas and mesenteric glands normal. Spleen 6 oz.; in all respects like a heart spleen, firm and dark coloured. Kidneys 11 oz.; the vessels were decidedly thick like those of a case of Bright's disease, and the organs were dilated like heart organs, but except for this they were quite healthy. Capsules thin, surfaces smooth, cortex in good quantity. Under the microscope the kidneys appeared perfectly healthy, but the arteries somewhat thickened, especially their adventitia. The liver showed well-marked interstitial fibrillation, with but little small-celled growth, and much fibro-hyaline exudation. The spinal cord did not exhibit any marked changes, beyond slight thickening of the adventitia of the vessels. Both great toe-joints contained a little urate of soda. The knees showed senile wasting of the central part of the cartilage. I noticed nothing wrong with the tendinous structures around the joint, nor in the ligamentum patellæ."

CASE 32. *Chronic renal dropsy; very high arterial pressure; extensive retinitis; urine normal.*—Frederick B—, æt. 47. Came under my care as an out-patient on March 21st, 1881. A tanner. Father died of some surgical affection; mother dead, cause unknown. He had "fits" fifteen years ago. He has been ill more or less since 1869, he then had what he calls "inflammation of the brain." He has had many abscesses in the head, neck, groins, &c. Was in Naaman ward in 1877; he was then suffering from syphilitic otitis of the temporal bone. He had puffiness of the face in connection with the local disease, and appears never to have lost it. His urine was always free from albumen, whenever examined on this occasion.

He has a typical renal appearance. Intense anæmia. Great œdema of the face and limbs; the œdema not pitting readily, especially about legs, where it is hard and brawny to the touch. The apex beat cannot be felt, the sounds are indistinct. His pulse is one of very high pressure as shown both by the finger and the sphygmograph.



Pressure 3 oz.

There is very extensive retinitis of the right eye, and much atrophy of choroid. His urine contained no albumen. Again, on March 28th, it is noted that his urine is still free from albumen.

CASE 33. *Two attacks of renal dropsy without albuminuria; very high arterial pressure; urine normal.*—George D—, æt. 53. A cellarman. Came under my care as an out-patient in August, 1880. He has generally had good health, has never drunk freely, about a pint and a half of beer a day. On this first occasion he had typical renal dropsy, but his urine was found to be free from albumen. At that time he rapidly improved and his dropsy went in about a fortnight. On January 17th, 1881, he again attended. His dropsy had been coming on for a fortnight; the weather had been bitterly cold. He again presented typical renal dropsy; face, legs, arms, all

highly œdematous. Some little cough. Apex beat cannot be felt. Second sound highly accentuated. Pulse of high pressure, hard, and long. Sphygmographic tracing taken, 6 oz. of pressure required and very high pressure tracing obtained. He has never had gout. Atropine was used to fully dilate his pupils, and the fundus was found to be normal and free from hæmorrhages. Urine faintly acid, sp. gr. 1012, no albumen. Faint cloud on heating, immediately soluble in acetic acid. No cloud with nitric acid. On my expressing my surprise to the students, and drawing their attention to the fact, he remarked, with some interest "That's what you said before!"

CASE 34. *Acute renal dropsy; high arterial pressure; hypertrophied heart; urine normal.*—Joseph R—, æt. 43. Dr. Hilton Fagge (Clin. clerk, Mr. Griffiths). October 1st—October 18th, 1879. A hawker; has drank freely. Family history good as far as it is known. No previous illness. His present illness began on September 19th (nearly a fortnight ago); he then noticed his feet were swollen; he took no heed of it for ten days when he came to the hospital, as he found his feet and legs getting gradually larger; the swelling beginning in the feet and extending gradually upwards. He is a short, stoutly built man, with the puffy appearance of renal dropsy. He feels perfectly well in himself. There is general œdema of face, body, and extremities, all well marked, but not to an excessive degree. There is a "renal cushion" well developed over the sacrum. Heart's apex in fifth interspace and in nipple line. Arteries feel thick. Pulse long, hard, and persistent. The sphygmographic tracing shows very high arterial pressure.



Pressure 7 oz.

The urine is clear and free from albumen, at first of low specific gravity and of excessive quantity. The œdema soon disappeared and the pulse showed a reduction of arterial pressure. He went out quite well.

Date.	Quantity.	Sp. gr.	Solids.	Alb.
Oct. 2	—	1010	—	None.
3	75 oz.	1012	900	„
4	80 „	1012	960	„
5	88 „	1012	1056	„
6	92 „	1015	1380	„
7	98 „	1012	1776	„
8	80 „	1018	1440	„
9	90 „	1017	1530	„
10	90 „	1020	1800	„
11	92 „	1019	1748	„
12	98 „	—	—	„
13	64 „	1019	1216	„
14	92 „	1019	1748	„
15	68 „	1020	1360	„
16	78 „	1020	1560	„
17	40 „	1023	920	„

CASE 35. *Acute renal congestion with renal dropsy ; faint trace of albumen for two days ; hypertrophied heart ; thick arteries ; high arterial pressure.*—William T—, æt. 43. Dr. Moxon (Clin. clerk, Mr. Duckworth). Admitted October 14th, 1880. Discharged October 31st, 1880. He is a corn porter, and has always drank heavily, many pints of beer and several glasses of rum a day. No serious illnesses. Nine days before admission he felt cramping pains across his loins. His scrotum, penis and ankles became swollen. He noticed that he passed less water than usual. On admission he is a big, bloated-looking man. His conjunctivæ, face, legs, ankles, scrotum, and penis are all œdematous. There is a little fluid in the peritoneal cavity. There is crepitation at the bases of both lungs behind. The heart is somewhat enlarged ; the area of dulness is increased. Apex beat indistinct. First sound prolonged ; there is an occasional systolic bruit. Arteries somewhat thickened. Pulse persistent, small, and compressible. The liver is enlarged, reaching two finger's breadths below ribs. Spleen normal. Tongue fairly clean. Bowels regular. Appetite good. The urine is pale, sp. gr. 1020, contains a trace of albumen. October 15th.—Anasarca less general. Only a trace of albumen in urine. October 16th.—The dropsy of trunk, face, scrotum, penis, &c., has disappeared. Patient looks well. Appetite is good. Bowels regular. The urine is free from albumen. After this the

albumen never again appeared, and he was discharged on October 31st apparently well, though the history of alcoholic excesses, his bloated, unhealthy appearance, the enlargement of his heart, and the thickness of his vessels all give evidence of the chronic nature of his renal disease.

Date.	Quantity.	Sp. gr.	Solids.	Alb.
Oct. 14	...	1020	...	A trace.
15	...	1020	...	"
16	84 oz.	1010	840	None.
17	132 "	—
18	40 "	1016	640	None.
19	60 "	1020	1200	"
20	52 "	1020	1040	"
21	60 "	1024	1440	"
22	52 "	1023	1196	"
23	40 "	1022	880	"
24	40 "	—	—	—
25	40 "	1022	880	None.
26	40 "	1020	800	"
27	36 "	1020	720	"
28	40 "	1020	800	"
29	32 "	1020	640	"

CASE 36. *General œdema; hypertrophied heart; high arterial pressure; urine normal.*—W. H. C—, æt. 46. Dr. Moxon (Clin. clerk, Mr. Rowlands). July 14th—August 4th, 1880. He is said to have been more or less troubled since childhood with some eye affection and headache, otherwise has had tolerably good health. Twenty-six years ago he had gonorrhœa, a soft chancre, and bubo, with a slight sore but no rash. About five months ago the sight in both eyes began to fail, and he had a slight brow-ache at the same time; did not improve under treatment, but sight has been getting worse up to admission. For the last fortnight he thinks he has been losing power in his legs and hands. He is an anæmic-looking man, with a puffy face and dark hair and eyes. Complains of slight pain in the head and a sense of heaviness over lower part of chest. He is said to have want of power in hands and legs; he seems to stagger a little in walking. He complains of a slight sense of numbness in his feet. There is no definite paralysis, but there is a lack of

energy about all his movements ; this has been coming on a fortnight or so. There is no anæsthesia. Patellar tendon-reflex very slight. He is nearly blind. These are all the symptoms of nervous disease. On the other hand, he has well-marked general œdema ; his face is distinctly puffy ; there is sub-conjunctival œdema, œdema of the thorax, feet, and legs ; behind there is a very distinct "renal cushion." The apex beat of the heart can only be felt with difficulty ; the impulse is decidedly external to nipple ; the second sound is highly accentuated. Pulse long, persistent, and compressed with difficulty. The tracing obtained is highly characteristic of Bright's disease with very high arterial pressure.



Pressure 4 oz.

His optic discs are pale and watery looking, as though œdematous. There are no retinal hæmorrhages. His eyes were examined by Mr. Higgins, who reported as follows :— "Doubtful perception of shadows ; slight convergent strabismus, apparently from weakness of external recti. Optic discs pale ; outlines ill-defined." He remained in the hospital three weeks, without any material change in his symptoms. On August 3rd, the day before his discharge, the following note is made :—"Patient is stronger on his legs. Feet and legs more œdematous. Does not complain of any subjective sensations this week. His urine was examined daily. It was clear, amber coloured, it never contained albumen, nor could any casts be discovered."

Date.		Quantity.		Sp. gr.		Solids.		Bowels acted.
July 16	...	40 oz.	...	1020	...	800	...	1
17	...	40 "	...	1020	...	800	...	2
18	...	36 "	...	1020	...	720	...	2
19	...	38 "	...	1020	...	760	...	2
20	...	40 "	...	1020	...	800	...	1
21	...	32 "	...	1018	...	576	...	2
22	...	40 "	...	1020	...	800	...	2

Date.		Quantity		Sp. gr.		Solids.		Bowels acted.
July 23	...	28 oz.	...	1018	...	504	...	2
24	...	32 "	...	1018	...	576	...	1
25	...	16 "	...	1012	...	192	...	1
26	...	32 "	...	1016	...	512	...	1
27	...	24 "	...	1012	...	288	...	1
28	...	44 "	...	1012	...	528	...	1
29	...	40 "	...	1012	...	480	...	1
30	...	40 "	...	1012	...	480	...	1
31	...	20 "	...	1022	...	440	...	1
Aug. 1	...	22 "	...	1022	...	484	...	1
2	...	30 "	...	1016	...	480	...	1
3	...	40 "	...	1012	...	480	...	1

CASE 37.—*Chronic bronchitis and emphysema ; general œdema ; high arterial pressure ; degenerate vessels ; urine normal.*—John D—, æt. 43. Under Dr. Pavy (Clin. clerk, Mr. Lawson). January 25th—April 11th, 1880. Has suffered from cough for three or four years, which has become worse lately. There has occasionally been bright blood mixed with sputa, especially in the spring time. Has noticed that his breath smelt badly during the last two or three months. He has had dyspnœa for two years, but this has been much worse for the last six weeks. On admission, he has a puffy and cyanosed face, much subconjunctival œdema, œdema of legs and trunk, a well-marked renal cushion, and some ascites. Hands wasted, and fingers clubbed by prolonged cyanosis. Liver enlarged and tender. His breath and sputa are extremely offensive. Chest emphysematous in shape and hyper-resonant. Bronchitic sounds throughout both lungs ; small crepitations at both bases. Heart's impulse felt in epigastrium and diffused over a large area ; apex cannot be localised ; loud and accentuated second sound, feeble first. Very thick and degenerate arteries ; the radials are tortuous and beaded. Pulse incompressible. Gets up two or three times at night to micturate. Urine, sp. gr. 1015, no albumen. March 3rd.—Lips purple. Heart's action intermittent. No albumen in urine. March 4th.—Urine, sp. gr. 1025, no albumen. March 7th and 8th.—No albumen. The further record of urine appears in the following table.

Date.	Quantity.	Sp. gr.	Solids.	Alb.	Bowels.
—	—	—	—	—	—
Mar. 10	... 40 oz.	... 1030	... 1200	... None	... Open.
11	... 40 "	... 1030	... 1200	... "	... "
12	... 62 "	... 1025	... 1550	... "	... "
13	... 46 "	... 1025	... 1150	... "	... "
14	... 50 "	... 1025	... 1250	... "	... "
15	... 54 "	... 1020	... 1080	... "	... "
16	... 50 "	... 1030	... 1500	... "	... "
17	... 50 "	... 1020	... 1200	... "	... "
18	... 62 "	... 1020	... 1240	... "	... "
19	... 64 "	... 1020	... 1280	... "	... "
20	... 60 "	... 1020	... 1200	... "	... "
21	... 58 "	... 1020	... 1160	... "	... "
22	... 61 "	... 1020	... 1220	... "	... "
23	... 70 "	... 1020	... 1400	... "	... "
24	... 66 "	... 1025	... 1650	... "	... "
25	... 62 "	... 1028	... 1736	... "	... "

During his stay in hospital he was troubled by being able to pass only a small quantity of urine at a time, only three or four ounces. His heart, on several occasions, was very irregular. When he left, on April 11th, he was still very ill.

CASE 38.—*Chronic bronchitis and emphysema ; renal dropsy ; rather high pressure ; albuminuria on three occasions, usually normal urine.*—Louisa L—, æt. 50. Dr. Fagge (Clin. clerk, Mr. Perks). January 11th—February 5th, 1881. Family history uncertain. She has had winter cough for many years. About one year ago she was laid up with swelling of chest and abdomen. Was better during the summer, but a day or two after Christmas she was again seized with cough, difficulty of breathing, and swelling of feet, face, and hands. An extremely fat, flabby woman, accustomed to drink gin, at least two glasses a day ; superficial vessels of face and of conjunctiva dilated and varicose. Œdema of conjunctiva, feet, and hands. A small amount of ascites. There was harsh breathing, prolonged expiration, a few moist sounds, with sibilant rhonchi all over chest. The apex beat was in fifth interspace, about three inches from sternum. Cardiac dulness normal. No visible pulsation. First sound distant, heard best at apex, very distant over lower extremity of sternum. Second sound sharp and accentuated, especially at apex. No bruit. Pulse regular,

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rather long, hard, not very compressible. Pulse tracing, taken on January 18th, indicates blood pressure but little above the



average. Liver not enlarged. Urine was found to contain albumen, which disappeared the next day, and was only present on two subsequent occasions.

Date.	Quantity.	Sp. gr.	Solids.	Alb.	Bowels.
Jan. 13	... 6 oz. (P) ...	1020	... 664	... $\frac{1}{2}$... 3
14	... 37 „	1018	... 666	... None	... 1
15	... 43 „	1018	... 774	... „	... 1
16	... 46 „	1020	... 920	... „	... 1
17	... 36 „	1016	... 476	... „	... 1
18	... 12 „	1015	... 330	... „	... 1
19	... 36 „	1018	... 648	... „	... 1
20	... 29 „	1016	... 464	... „	... 1
21	... 26 „	1015	... 390	... „	... 1
22	... 38 „	1015	... 570	... „	... 1
23	... 31 „	1018	... 568	... „	... 1
24	... 28 „	1016	... 448	... „	... 1
25	... 27 „	1018	... 486	... „	... 1
26	... 24 „	1020	... 480	... „	... 1
27	... 28 „	1018	... 524	... „	... 1
28	... 28 „	1018	... 524	... „	... 1
29	... 36 „	Got up to-day	—	... „	... 2
30	... 20 „	1020	... 400	... $\frac{1}{10}$... 1
31	... 32 „	1011	... 576	... None	... 1
Feb. 1	... 28 „	1022	... 616	... „	... 1
2	... —	1024 „	... 1
3	... —	1020 „	... 2
4	... —	1018 „	... 1
5	... —	1020 Slight cloud	... 1
6	... —	1018 None	... 1
7	... —	1022 „	... 1

Exception may, perhaps, be taken to this case ; its nature is certainly not well established, neither hypertrophy of the heart nor high arterial pressure being demonstrated ; yet the

well-marked renal dropsy and the presence of albumen on three occasions have been taken as sufficient evidence to warrant its being classed in this group.

CASE 39. *Epileptiform attack; uræmia (?) ; hypertrophied and dilated heart; high arterial pressure; slight general œdema; urine normal.*—Frederick F—, æt. 56. Dr. Pye-Smith (Clin. clerk, Mr. Hind). December 7th—December 29th, 1879. A cabdriver. Family history good. He says that he has been subject to epileptic fits as long as he can remember, for the last few years having one about every two years. When an attack comes on he first feels dizzy and then becomes unconscious; is generally ill five or six weeks after each attack; says he has drunk heavily years ago, both beer and spirits. On the night of December 6th was walking over Blackfriars Bridge when suddenly he felt dizzy and fell down; knows nothing more until he found himself in Guy's. I saw him the next day, and made the following note:—"He looks like a man who has drunk heavily, his face is red and bloated; he has the appearance of one suffering from so-called 'uræmia'; he is semi-conscious and muddled, his movements are slow and feeble; he is lying low in his bed and on his back. When asked a question he makes some absurd answer, rambling on in an incoherent manner; he can only be roused with difficulty to make even this effort. His pupils are contracted, and do not react to variations in light. There is well-marked subconjunctival œdema, general puffiness of the face, especially of the lower eyelids; there is slight œdema of the hands and legs. He has an ammoniacal smell. Pulse typically that of chronic Bright's disease, 75, regular, very persistent, incompressible, and long; artery thickened. Heart's apex beat greatly displaced, faintly felt below sixth rib and $1\frac{1}{2}$ inches external to nipple; systole short, like that of dilatation." Urine, sp. gr. 1025, clear, reaction slightly acid, contains no blood, albumen, or sugar. Temp. $98\cdot4^{\circ}$.

December 10th.—Complains of pain in his head; seems very drowsy. Dec. 11th.—Urine 1025, no albumen or blood. Seems a little better. Dec. 12th.—More sensible, and answers questions rightly. Still complains of pain in his

head. Dec. 14th.—Urine healthy. Has no œdema of hands or legs. Dec. 16th.—Urine 1080, clear, no albumen, blood, or sugar. No œdema of legs. Dec. 24th.—Heart sounds have a somewhat tic-tac character; second sound accentuated at base. Coarse breathing, long expiration, and some rhonchus scattered all over chest. Dec. 27th and 29th.—Urine, sp. gr. 1020 and 1015, no albumen. He left on the latter day, still complaining, however, of pain in his head.

V. CASES WITH GOUT.

CASE 40.—*Plumbism; gout; renal dropsy; high arterial pressure; transient mitral regurgitation; urine normal.*—Robert D—, æt. 36. Dr. Pye-Smith (Clin. clerk, Mr. Gardner). August 13th, 1879—January 20th, 1880. A shoemaker, no reason for the lead poisoning discovered, beyond a habit of putting nails into his mouth when rivetting, and the fact that the leather is cut on lead before it comes into his hands. He has been a hard drinker. He was more or less unwell in November, December, and January, during which months he had "colds," pains in his back and limbs, and a short pyrexial attack. Towards the end of January the fingers of his right hand became useless and his right wrist dropped. During the last four or six weeks his left wrist also became affected. On admission he is suffering from paralysis of the extensors of both forearms; he is anæmic, has a well-marked blue line on gums. His joints are unaffected. The second sound of the heart is loud and sharp, and can be heard all over the right side of chest. The clerk describes his pulse as "very weak," but I have no special note about its characters. Although the urine was saved and measured daily no notes of its characters are recorded. I believe that it may safely be assumed to have been normal as my attention had been especially directed to the fact at the time. During August and September his paralysis slowly but steadily improved under the use of the continuous current applied daily.

October 11th.—Has been taken ill to-day with sickness and diarrhœa after eating a quantity of grapes.

13th.—Says he feels very ill this morning, complains of

constant sickness and diarrhoea. Temperature 96·8°. Pulse 108, strong, regular, and incompressible.

16th.—Is all right again this morning.

17th.—This morning he is lying in bed with the scrotum greatly swollen and œdematous. The legs are also slightly œdematous. No albumen in urine. Slight bruit heard at apex.

22nd.—I note that—"He looks intensely renal. A pale, pasty, and œdematous-looking face. A 'renal cushion' over sacrum; scrotum and legs œdematous. Urine, no albumen, uric acid deposit. Pulse persistent, but not long. Apex beat perceptible, but not displaced, first sound inaudible at apex, replaced by a soft systolic murmur; second, intense and tympanitic at apex, more so than at base." The urine was saved, measured and tested daily from now till November 16th. He steadily improved.

November 6th.—Feels much better now, but still complains of cramp in his fingers and toes. *No bruit* can be heard at apex now; the urine contains no uric acid, and the legs are not œdematous.

On December 8th he had an attack of gout in his feet which laid him up for three days; this recurred on January 8th, after this he steadily improved, and when he left he had completely regained the power of the wrist.

The urine table appears to be very inaccurate as to the number of ounces passed, and it can only be relied on to represent a minimum quantity.

During the whole of December the quantity of urine passed is recorded, but its specific gravity only occasionally, when it is also noted that it contains no albumen.

Date.	Quantity.	Sp. gr.	Solids.	Alb.
Oct. 23	26 oz.	1025	700	None.
24	40 "	1020	800	—
25	24 "	1020	480	—
26	—	—	—	—
27	26 "	1020	520	None.
28	34 "	1020	680	"
29	—	—	—	—
30	34 "	1025	850	"
31	34 "	1025	850	"
Nov. 1	24 "	1025	600	"

Date.	Quantity.	Sp. gr.	Solids.	Alb.
Nov. 3	... 20 oz.	... 1025	... 500	... None.
4	... 40 "	... 1025	... 1000	... "
6	... 60 "	... 1025	... 1500	... "
10	... 28 "	... 1030	... 840	... "
11	... 32 "	... 1027	... 756	... "
13	... 20 "	... 1025	... 500	... "
14	... 30 "	... 1028	... 840	... "
15	... 20 "	... 1007	... 140	... "
16	... 12 "	... 1023	... 276	... "

CASE 41.—*Plumbism; gout; hypertrophied heart; mitral regurgitation; bronchitis; epistaxis; urine normal.*—Thos. M—, æt. 48. Dr. Wilks (Clin. clerk, Mr. W. Spong). January 31st—February 28th, 1880. A paperhanger and painter. Has never had scarlet or rheumatic fever. First attack of gout four years ago. Winter cough for last twelve years. Has drunk freely. He came in for cough associated with severe epistaxis. He is described as a sparely built man, fairly well nourished, medium height and weight. Somewhat anæmic, pasty face, well marked subconjunctival œdema, lower eyelids puffy. Hair dark, no beard or moustache. Eyesight good, sclerotics clear. No anasarca of lower extremities. Complains of sickness in the early morning with loss of appetite; eyes watery on rising; blue line on gums. (Has worked a good deal in lead, never had colic.) Extent of precordial dulness not increased. Apex beat can be felt half an inch below and in nipple line; systolic bruit can be heard at apex and there only, very faint and somewhat high pitched. Pulse regular and compressible. Unfortunately I have no actual note of my own about the pulse in this case, and the report does not state whether it was persistent or not. But my memory of the case is sufficiently clear to assert that it was one of high pressure. Has not passed much water lately. Urine light colour, clear, no deposits, sp. gr. 1022, no albumen, sugar or blood.

February 2nd.—Epistaxis again commenced severely.

3rd.—Is still troubled with epistaxis at times. Persistent headache. The bruit is more audible to-day.

16th.—No return of epistaxis since the 4th. Cough more troublesome, expectoration muco-purulent and streaked with

blood. Air said to enter left lung better than right; expiration prolonged at left apex. There was thought to be a little deficiency of resonance at this spot, but this was not confirmed by subsequent observations.

20th.—Bruit scarcely audible to-day.

24th.—Scarcely any cough or expectoration.

26th.—Gout in hand and foot, which subsided in a day or two. His urine never contained albumen. The evidence of vascular disease is not very satisfactory in this case, but his characteristic appearance and puffy face taken together with the fact that he had both gout and lead poisoning, make the diagnosis of granular kidneys and vascular changes very probable. He had no history of rheumatism to account for his mitral bruit, which was therefore probably due to leakage from an over-distended ventricle; the variable intensity of the bruit would seem to bear this out. His epistaxis and severe headache are characteristic of Bright's disease.

Date.	Quantity.	Sp. gr.	Solids.	Alb.
Feb. 4	16 oz.	1022	352	None.
5	12 "	1018	216	"
6	26 "	1018	468	"
7	30 "	1016	480	"
8	30 "	1017	510	"
9	36 "	1020	720	"
10	38 "	1018	684	"
11	20 "	1017	370	"
12	36 "	1018	648	"
13	38 "	1017	646	"
14	26 "	1016	416	"
15	36 "	1018	648	"
16	32 "	1020	640	"
17	—	1020	—	"
18	30 "	1022	660	"
19	40 "	1020	800	"
20	36 "	1015	540	"
21	32 "	1020	640	"
22	40 "	1020	800	"
23	34 "	1022	748	"
24	26 "	1023	598	"
25	38 "	1022	836	"
26	30 "	1020	600	"
27	26 "	1020	520	"
28	46 "	1020	920	"

CASE 42. *Gout ; hypertrophied heart ; thick vessels ; high arterial pressure ; transient albuminuria.*—William M—, æt. 45. Dr. Moxon (Clin. clerk, Mr. Trott). September 18th—October 4th, 1879. Father dead, cause unknown. Mother died of dropsy. One sister rheumatic. Four years ago patient had "rheumatism" in both ankles and slightly in elbows and shoulders. He now complains of severe pains in great toes and swelling of legs, with pains in several other joints. Never had crackling in joints. A strong-looking man with capillary injection of cheeks. There appears to be some bronchitis. The apex of the heart beats two inches below nipple, first sound at apex long, second sharp and accented. Pulse incompressible, full, and persistent. Arteries thick. There is no account of the condition of the joints but the disease was diagnosed as gout ; it was treated and relieved by colchicum. The condition of the urine is recorded as follows :

Date.	Quantity.	Sp. gr.	Solids.	Alb.
Sept. 16	34 oz.	1019	646	Some.
17	20 "	1020	400	
19	20 "	—	—	A little.
20	16 "	1020	320	
21	13 "	—	—	
22	20 "	1020	400	Slight trace.
23	34 "	—	—	
24	36 "	—	—	
25	40 "	1020	800	No alb.
26	52 oz.	—	—	None.
27	38 "	—	—	
28	38 "	1018	684	"
29	40 "	1016	640	"
30	44 "	—	—	
Oct. 1	60 "	—	—	
2	50 "	1010	500	"
3	40 "	1016	640	"

CASE 43. *Gout ; bronchitis ; hypertrophied heart ; high arterial pressure ; urine normal.*—Charlotte F—, æt. 61. Dr. Taylor (Clin. clerk, Mr. J. B. Howell). January 21st—March 30th, 1881. She was admitted into the hospital for bronchitis and chronic gout, she has been in twice before with the same complaints. Has had no other serious illnesses.

She is a well-nourished woman, with a slightly flushed face, and some dyspnoea. She has well-marked gout in the right hand and both feet, and a good deal of bronchitis, rhonchus and mucous râles being scattered over the chest. The apex of the heart can be felt one and a half inch below and in a vertical line with the nipple. Præcordial dulness not increased. Heart sounds normal. Urine very pale, sp. gr. 1015, acid reaction, slight precipitate on boiling, dissolved immediately by nitric acid. She passed through a severe attack of gout, which attacked several joints and therefore gave rise to a suspicion of the disease being rheumatism. During March she had an attack of severe abdominal pain, which appeared to be caused by some cæcal troubles. The accompanying tracing obtained from her pulse in the early part of March affords good evidence of her very high arterial pressure.



Pressure 5 oz.

Her urine was carefully watched during the month of March and appears to have been always normal. The quantity of urine saved is very deficient, this was probably due to want of care in preserving it, a difficulty more especially felt in the female wards.

Date.	Quantity.	Sp. gr.	Solids.	Alb.	Bowels.
Mar. 5	24 oz.	1022	528	None	2
6	24 „	—	—	„	„
7	20 „	1020	400	„	1
8	18 „	1020	360	„	1
9	22 „	1020	440	„	1
10	32 „	1015	490	„	1
11	28 „	1015	—	„	1
12	30 „	1015	450	„	„
13	—	—	—	„	„
14	32 „	1020	640	„	1
15	34 „	1020	680	„	1
16	30 „	1015	480	„	1

Date.		Quantity.		Sp. gr.		Solida.		Alb.		Bowels.
Mar. 17	...	34 oz.	...	1012	...	408	...	None.	...	1
18	...	28 "	...	1015	...	420	...	"	...	1
19	...	32 "	...	1015	...	480	...	"	...	1
20	...	36 "	...	—	...	—	...	"	...	0
21	...	32 "	...	1010	...	320	...	"	...	1
22	...	38 "	...	1012	...	456	...	"	...	1
23	...	38 "	...	1012	...	456	...	"	...	1

CASE 44. *Osteo-arthritis (gouty); hypertrophied heart; high arterial pressure; remarkable family history of apoplexy; urine normal.*—Sarah L—, æt. 51. Dr. Wilks (Clin. clerk, Mr. W. Spong). January 2nd—February 23rd, 1880. Her mother was subject to "rheumatism;" she had seven children, and died of apoplexy when thirty-nine. Patient is the only surviving child. Two sisters and one brother died of apoplexy, aged respectively fifty-one, thirty-one, and fifty. The three other children died when quite young. The patient has always had good health until sixteen years ago, when she was first troubled with what she calls rheumatic pains in most of her joints. For the last year or so she has been gradually getting more crippled, and has lost flesh considerably. Four years ago she says she had inflammation of the lungs, and was confined to her bed for three months. For the last few years she has had winter cough. For three weeks before admission she has been confined to her bed with pain in various joints. She has a pale, anæmic, pasty face; puffy lower eyelids, much subconjunctival cedema. Very many of her joints are severely and characteristically distorted by osteo-arthritis. There are some signs of bronchitis in the chest. Apex beat forcible and heaving, it is somewhat displaced, it is felt best in the fifth space and in line with the nipple. Cardiac dulness not increased. No bruit audible; second sound intensely accentuated. Pulse 84, characteristic of very high arterial pressure; arteries extremely thick. Urine light in colour, no deposits, and no albumen. The urine chart appended shows that its specific gravity was usually low, commonly only 1010, its quantity was rather increased if anything, it never contained a trace of albumen. She went out much as she came in.

Date.	Quantity.	Sp. gr.	Solids.	Alb.
Jan. 11	50 oz.	1012	600	None.
12	48 "	1012	376	"
13	20 "	1020	400	"
14	22 "	1016	352	"
15	22 "	1016	352	"
16	40 "	1017	680	"
17	—	1015	—	"
18	56 "	1012	672	"
19	38 "	1014	532	"
20	20 "	1014	280	"
21	28 "	1012	336	"
22	30 "	1012	360	"
23	28 "	1012	336	"
24	20 "	1010	200	"
25	22 "	1012	264	"
26	23 "	1014	322	"
27	60 "	1010	600	"
28	40 "	1012	480	"
29	44 "	1012	528	"
30	20 "	1020	400	"
31	—	—	—	"
Feb. 1	46 "	1012	552	"
2	28 "	1010	280	"
3	30 "	1010	300	"
4	60 "	1010	600	"
5	40 "	1012	480	"
6	38 "	1012	456	"
7	40 "	1014	560	"
8	60 "	1010	600	"
9	48 "	—	—	"
10	48 "	1012	576	"
11	56 "	1010	560	"
12	40 "	1010	400	"
13	50 "	1010	500	"
14	54 "	1011	594	"
15	60 "	1010	600	"
16	—	—	—	"
17	46 "	1012	552	"
18	48 "	1012	576	"
19	—	—	—	"
20	40 "	1012	480	"
21	60 "	1010	600	"
22	24 "	1012	288	"
23	30 "	1015	450	"

arterial pressure ; urine normal.—Robert D—, æt. 56. Dr. Pavy (Clin. clerk, Mr. Bolton). July 3rd—September 3rd, 1880. Subsequently under my own care as an out-patient. His father died of “gout in the stomach.” He has been subject to attacks of gout since he was twenty-eight years old, if not before. He was in Guy’s Hospital in 1877, suffering from gout. His urine was then normal. He is a tall, well-made man, but his joints are greatly deformed by gout. He has œdema of his face, legs, and scrotum, and it was this that caused him to seek admission. He has considerable dyspnœa on exertion. Has to rise many times at night to micturate. Apex beat $2\frac{1}{2}$ inches below nipple and one inch external to mammary line ; first sound normal, second intensely tympanitic, heard widely all over the chest. Pulse persistent and long. Artery is not recognisably thickened. Urine free from albumen. July 9th.—The urine is carefully examined daily for albumen ; there is no trace of it. July 15th.—No trace of albumen ; urine passed last twenty-four hours, 44 oz. Unfortunately, no complete record of the urine was preserved. The patient came under my care as an out-patient on January 31st, 1881. He had then no œdema ; his urine was free from albumen ; he had great hypertrophy of the heart. Apex beat $1\frac{1}{2}$ inches external to nipple and about 1 inch below.

VI.. CASES WITH SEVERE EPISTAXIS.

CASE 46. Severe epistaxis ; lumbar pain ; puffy face ; very high arterial pressure ; occasional albuminuria.—Richard R—, æt. 43. Dr. Pavy (Clin. assistant, Mr. Udale). January 30th—February 7th, 1881. The following case did not come under my own personal observation, but its carefully recorded details makes it of great value. For the last five years the patient has been subject to attacks of rheumatic gout, *i.e.* severe pain and swelling, commencing in the big toe, afterwards appearing in the knee, hands, &c. Except during these attacks his legs and feet have never swelled. Has suffered at times from an aching pain in the lumbar region. Sometimes his eyes get puffy. Has vomited in the morning on getting up, about once or twice a week, though some weeks not at all.

Some palpitation of the heart on exertion but not enough to cause much distress. After reading for some time his sight becomes hazy. Now and then he feels giddy, and his head swims. Frequent micturition, but little at a time.

January 27th, while at work, his nose began to bleed and continued for some time; this was repeated on the following day. On the 29th the bleeding was so profuse that he called in a medical man who plugged the anterior nares; this did not quite stop the bleeding so he applied to the hospital. When brought here he was very pale, almost pulseless, and in a cold sweat. He brought up a quantity of blood which he had swallowed, and while in the surgery he fainted. The patient is a strong, well-built man. Skin warm, dry, and anæmic. Conjunctivæ slightly œdematous. Slight effusion into left knee. No œdema of legs, &c. Slight rhonchus on deep inspiration, otherwise the lungs are healthy. Apex beat in normal position, no increase of cardiac dulness. A soft systolic bruit is heard indistinctly at the apex, which is more distinct at the base and on the right side of the sternum. Pulse strong, full, but compressible, very persistent, feeling like a cord rolling beneath one's fingers. Pulse regular.



Pressure 5 oz.

Urine rather dark amber coloured, acid, sp. gr. 1012, cloud of albumen on boiling, no sugar, about seven grains of urea to the ounce.

Date.	Quantity.	Sp. gr.	Solids.	Urea.	Albumen.
Jan. 30	...	1012	...	7 grs. to oz.	A trace.
31	...	—	...	—	Slight.
Feb. 1	...	—	...	—	Very slight.
2	...	—	...	—	"
3	43 oz.	1015	645	7.92 per oz.	None.
4	51 "	—	...	—	"
5	32 "	—	...	7.48 per oz.	...
6	51 "	1013	663	5.62 "	Slight trace with heat, none with nitric acid.

CASE 47. *Severe epistaxis ; chronic bronchitis and emphysema ; thickened and degenerate vessels ; urine normal.*—John P—, æt. 54. Dr. Pye-Smith (Clin clerk, Mr. Gosse). November 29th, 1878—January 9th, 1879. A builder's labourer. Family history unknown. Has always been a very heavy drinker, more whisky than beer. Up to five years ago he enjoyed good health, except for a continued fever twenty-seven years ago. Has never had rheumatism. Five years ago he had a fall on his left side and broke three ribs ; he was laid up for five weeks, and then went to work again ; has always had slight pain in his side since. Four years ago was his next illness, when coming home from work one evening he had violent epistaxis ; it lasted for five or six hours. A doctor was sent for who plugged the anterior nares, but then it came through his mouth. He says he lost two or three quarts of blood, which was of a very dark colour ; before the epistaxis he had vertigo and says he felt stupid. After this he was extremely weak, and was laid up for three weeks ; he had no more hæmorrhage. He has had more or less cough ever since this time. Three months ago his cough became more severe and his breathing difficult at night. Two months ago he had epistaxis again, in the night ; he had it every night for three weeks ; he says the attacks lasted about an hour, during which time the bleeding was very profuse. He says he has sometimes had pain in his loins. He sweats a great deal at night, and his chief complaints are cough and weakness. On admission, he is a well-made man, about the ordinary height. He has an anxious expression. Skin intensely anæmic, hot, and moist ; lips very pale. Conjunctivæ are a little jaundiced, there is well-marked sub-conjunctival œdema. No anasarca. His cough is very troublesome, and he expectorates much mucus but no blood. Breathing chiefly abdominal, resonance good all over, except for some impairment at right base behind ; expiration is rather prolonged, and there are dry râles all over right lung in front and behind. Impulse of heart cannot be felt. Præcordial dulness very indistinct, it is almost entirely hidden by highly emphysematous lung. Heart sounds very distant, second sound rather accentuated ; no bruit audible. Radial artery is very tortuous, it is quite visible. Pulse 64, slow, persistent,

and incompressible. Appetite fair, bowels usually regular. Liver dulness seems to be decreased; it begins at fifth space and only extends two inches in nipple line. Spleen not enlarged. Urine normal in colour and quantity, acid reaction, sp. gr. 1026, no albumen nor sugar. December 6th.—He complains of severe headache on left side of head. Dec. 7th.—Head still very bad; he had a bad night. Dec. 9th.—Head better this morning; he has had a slight epistaxis. Dec. 12th.—Headache continues; he has had some slight epistaxis during the night, which has relieved the head a little. After this he steadily improved up to the day of his discharge, on January 7th; he still remained very anæmic. His urine was always free from albumen; the notes are as follows :

Dec. 1.—No albumen.

3.	„	60 ounces.	
4	„	50	„
5.	„	54	„
6.	„	52	„
7.	„	60	„ Sp. gr. 1025.
15.	„	—	„ 1025.

During this time he was taking *Mistura Senegæ*, and Chloral at night.

CASE 48. *Severe epistaxis; high arterial pressure; hypertrophy of heart? urine normal.*—John M—, æt. 46. Dr. Goodhart (Clin. clerk, Mr. Strachan). March 22nd—April 2nd, 1880. Patient has been a navvy all his life, chiefly engaged in tunnelling on railroads. No history of hereditary disease. No previous illness of importance. He has lost his sight for two years by cataract. Has been a free drinker both of spirits and beer. For the last two months he has found it necessary to get up several times during the night to empty his bladder. He was admitted on March 15th into the surgical wards for extremely severe epistaxis, for which, failing all other remedies, his posterior nares were eventually plugged. On the following day the plug was removed, and this was followed shortly by another excessively severe hæmorrhage, which necessitated further plugging. This being removed without recurrence, he was transferred to the medical

wards for further treatment. He is an emaciated man, of medium height, and much blanched by the recent loss of blood. The area of præcordial dulness is not increased; the apex beats in the fifth space, but nearly three inches below the nipple, "owing to apparent displacement of the latter (perhaps from wasting)," it is situated in the nipple line. Impulse diffused over a rather large area. The first sound is rather thick and prolonged at apex, the second is accentuated at base and accompanied by an occasional creak. Pulse 100, rapid, regular, short, persistent, not easily compressible. Artery very hard, can be followed for some inches above the wrist. All other physical signs were normal. His appetite had been poor for the last two or three weeks, and his bowels confined. Urine acid, of normal colour, no albumen nor sugar, sp. gr. 1024. Notwithstanding his recent severe hæmorrhage and his anæmic and emaciated condition, the signs of increased arterial pressure were so marked that Dr. Goodhart did not fear to treat him for it; he accordingly prescribed Pulv. Jalapæ Co. ʒij, every morning, Mist. Rosæ Laxativæ ʒj, ter die, and Pil. Ferri Redacti gr. v, ter die. Full diet, without beer. The result of this treatment was rapid improvement. March 23rd.—No further hæmorrhage has occurred. Patient feels better. Temporal arteries are tortuous and very visible. Urine, sp. gr. 1022, no albumen. March 25th.—Feels stronger. Bowels have been opened three times. Urine 30 oz., sp. gr. 1020, no albumen. Pulse quieter. March 27th.—Continues better. Pulse regular, 88. Heart sounds normal. April 2nd.—Having been up several days, and feeling well, he left the hospital. Still no albumen in urine.

VII. CASES WITH VARIOUS MEDICAL AND SURGICAL DISEASES.

CASE 49. *Disease of knee-joint; resection; amputation; recurrent hæmorrhage; hypertrophied heart; pericarditis; granular kidneys; transient albuminuria after catheterism for stricture; death.*—John M—, æt. 58. Mr. Howse (Clin. clerk, Mr. L. E. Shaw). Admitted February 27th—November 5th, 1880. He injured his left knee-joint six months before admission, and this becoming worse caused him to

apply at the hospital. He has drunk rather freely. Has had gonorrhœa more than once, and suffers from a stricture, which has never been treated. At the time of admission it was noted that his apex beat was half an inch outside of nipple and rather pronounced; the radial artery tortuous and very much thickened. Some difficulty occurred with his stricture, and catheters were used for the first few days after admission; the first note of the urine is on March 5th, it states that the urine contains some pus corpuscles and epithelium cells, sp. gr. 1080. March 8th.—Sp. gr. 1080, very little albumen, no sugar. March 9th.—Sp. gr. 1080, very little albumen, no sugar. The albumen on these occasions was probably due to the presence of pus. March 10th.—Sp. gr. 1025, no albumen. After this there is no further note of the condition of the urine until April 8th. There is no note of any catheterisation after March 22nd. His knee-joint was excised on April 6th; this was followed by a little oozing, but no hæmorrhage of importance. In addition to recent suppuration, old osteo-arthritic changes were found in the joint. April 8th.—Urine sp. gr. 1025, acid, no albumen, no sugar. April 14th.—No albumen nor sugar.

On April 17th it is stated that the pulse has been intermittent for two days, in other respects he is doing well; there is little or no fever; the pulse was normal again on the 20th. During July and August his general health appears to have been fairly good, especially considering the severe local trouble. On September 21st some sinuses about the old wound were laid open, they were rather extensive. On October 27th it was discovered that there was a considerable amount of dead bone about the lower end of the femur; on November 2nd Mr. Howse amputated through the lower third of the femur. It is noted on this day that the urine does not contain albumen, sp. gr. 1024. Previous to the operation Dr. Goodhart examined the patient and failed to find any evidence of organic disease. The amputation was complete at 2.15 p.m. About 4 p.m. hæmorrhage from the stump was noticed; it was discovered to come from the bone, and slightly also from the soft parts; about a quart of blood was lost. On the night of November 4th he awoke restless and complaining of the pain in the stump; at 2 a.m. he had an injection of morphia (he

had frequently had them before). He then fell asleep, but he breathed heavily all night and woke at 7 a.m. He was then quite sensible, but complaining of pain in the stump, breathing heavily and groaning. He died three hours afterwards. There was some question whether his death was not due to blood poisoning, and in relation to this it is noted that a patient in this ward had developed a severe attack of erysipelas on October 31st, and was removed on November 2nd; another patient, æt. 70, died on November 4th, from broncho-pneumonia after an operation for strangulated hernia.

Post mortem (by myself).—A well-formed, but rather emaciated body. Thin old adhesions scattered all over both pleuræ. Rather firm matting about region of pericardium. Lungs coarse and somewhat emphysematous; tubes contained frothy secretion. Both layers of the pericardium were covered completely by a thick layer of lymph, with the usual shaggy surface; this could be stripped off the heart, and was about $\frac{1}{16}$ th of an inch in thickness. Heart-19 oz.; the muscle was fairly good, except the part immediately below the pericardium, here the muscle was pale and apparently fatty; left auricle slightly thickened; left ventricle considerably hypertrophied; aortic and mitral valves thick and opaque, otherwise normal; right side normal; a little atheroma in the aorta. Arteries generally thickened. The abdominal viscera were all normal. Liver 64 oz.; spleen $7\frac{1}{2}$ oz. Kidneys $8\frac{1}{2}$ oz.; both organs were rather small, the capsules peeled fairly well, but the structure was exceedingly indistinct and "muddled" looking; cortex atrophied somewhat; vessels decidedly thickened. The microscopical appearances of the kidney are exhibited in Plate II, which was taken from this specimen; it shows slight interstitial thickening, more especially due to the thickening of the *membrana propria* of the tubules; the capsules of the Malpighian glomerules were also distinctly thickened, and some of the tufts were atrophied and more or less obliterated; there was decided thickening of the muscular coat, and some of the *intima* of the arterioles; the epithelium is granular and excessive in amount; some of the tubes contain casts. Testes healthy.

CASE 50. *Aneurism of popliteal; ligature of femoral; hyper-*

trophy of heart; normal urine; subsequent acute nephritis during convalescence; death.—Elias W—, æt. 42. Admitted November 17th, 1880; died February 17th, 1881. Under Mr. Bryant. Suffering from aneurism of popliteal artery. He has previously had fairly good health. He had no albuminuria at the time of admission. His aneurism was treated by compression, which failed, and then by ligature, which cured it. No general signs of vascular disease were detected during his lifetime. His urine was very frequently examined; it was generally of small quantity and high sp. gr., owing to restrictions in the quantity of fluid diet. It was always free from albumen until January 10th, 1881. He was convalescent and about to get up when he developed an attack of acute renal dropsy, and died in about five weeks. His urine was tested several times before the operation, and was always perfectly normal.

Post mortem (by Dr. Hilton Fagge).—The kidneys weighed $14\frac{1}{2}$ oz.; they were blurred, greyish, and slightly yellow. Heart weighed 14 oz.; left ventricle dilated and hypertrophied; in the pericardium there was 16 oz. of clear, straw-coloured fluid. A considerable quantity of fluid in the pleuræ. Lungs small, nearly airless, œdematous; liver $54\frac{1}{2}$ oz., indurated, pale, and somewhat fatty; spleen 7 oz., firm.

CASE 51. *Multiple cysticerci cellulosa; high arterial pressure; urine normal; hypertrophied heart; granular kidneys; death.*—Mary S—, æt. 64. Dr. Habershon (Clin. clerk, Mr. R. T. Jones). March 12th; died April 23rd, 1879. A charwoman, has had winter cough three years, and recently œdema of the legs. Admitted with signs of bronchitis, yellow conjunctivæ, and prolongation downwards of the edge of the liver. As she was anæmic and wasted, cancer of the liver was suspected, and it was thought that the existence of malignant disease was confirmed by the discovery of numerous small nodules under the skin of the arms, chest, and back. They were not adherent to the skin. The urine was of good specific gravity (1018), and contained no albumen, but the pulse was persistent and long, and led me to the diagnosis of Bright's disease.

Post mortem (by Dr. Hilton Fagge).—A remarkable case of
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multiple cysticerci cellulossæ in the subcutaneous tissues, the heart, and the brain; the details of this condition may be omitted here, as they have no bearing on the condition of her heart and kidneys. In the cerebellum there was slight but extensive capillary apoplexy, no doubt a result of the granular condition of the kidneys; no cysticerci were present in this part of the brain. Lungs very emphysematous indeed, and highly œdematous; much purulent mucus in tubes. Heart 12 oz.; mitral valve rather thick edge; some arteritis deformans. Liver and spleen healthy. Kidneys 9 oz., granular, rather pale, with wasted cortex and large hilus.

CASE 52.—*Sciatica; hypertrophied heart; high arterial pressure; urine normal.*—William K—, æt. 72. Dr. Moxon (Clin. clerk, Mr. Dowson). Admitted September 7th—discharged October 27th, 1880. His family history was good. He has been employed for the last thirty years in a brewery, and has during this period drunk beer freely. When 18 years of age he had an attack of rheumatic fever. He had sciatica for the first time rather more than ten years ago; he was then in this hospital twice for this disease; the last time he had lumbago as well, and for this he was cupped over the loins, of which he still bears the marks. He has had no severe attacks for ten years, but has had occasional darting pains down the right leg. For some months he has suffered from headache and slight sickness in the early part of the day. He was admitted with severe and very typical sciatica, of which I need not give any further account. He is a dull, heavy-looking man, rather anæmic, his skin having a yellowish tinge. He has several sebaceous tumours about his body. His hair and eyebrows are very coarse and turning grey. There is no œdema. He has slight bronchitis and rhonchi are audible in the larger tubes. The apex of the heart beats in the sixth space half an inch external to the nipple line. The heart sounds are indistinct; the first is scarcely audible at base, but the second is accentuated. The cardiac dulness is increased. Pulse very long and persistent, but the artery is little if at all thickened. His sciatica was much relieved by treatment; in other respects he went out as he came in. His urine was very carefully watched throughout, and the table

appended shows its uniformly normal quality and quantity. It was invariably acid and free from albumen, for which it was tested daily. Uric acid crystals were present in it on two occasions.

Date.		Quantity.		Sp. gr.		Solids.
Sept. 9	...	32 oz.	...	1020	...	640
10	...	52 "	...	1018	...	936
11	...	32 "	...	1022	...	704
12	...	56 "	...	—	...	—
13	...	60 "	...	1014	...	840
14	...	80 "	...	1010	...	800
15	...	68 "	...	1010	...	680
16	...	68 "	...	1010	...	680
17	...	64 "	...	1010	...	640
18	...	56 "	...	1012	...	672
19	...	40 "	...	—	...	—
20	...	40 "	...	1011	...	440
21	...	68 "	...	1012	...	816
22	...	56 "	...	1014	...	784
23	...	40 "	...	1020	...	800
24	...	40 "	...	1018	...	720
25	...	60 "	...	1012	...	720
26	...	—	...	—	...	—
27	...	52 "	...	1012	...	624
28	...	48 "	...	1012	...	576
29	...	40 "	...	1020	...	800
30	...	60 "	...	1012	...	720
Oct. 1	...	40 "	...	1012	...	480
2	...	32 "	...	1012	...	384
3	...	—	...	—	...	—
4	...	32 "	...	1022	...	704
5	...	80 "	...	1012	...	960
6	...	—	...	—	...	—
7	...	—	...	—	...	—
8	...	40 "	...	1018	...	720
9	...	44 "	...	1018	...	792
10	...	—	...	—	...	—
11	...	32 "	...	1020	...	640
12	...	60 "	...	1010	...	600
13	...	40 "	...	1015	...	660
14	...	48 "	...	1012	...	576
15	...	48 "	...	1014	...	672
16	...	60 "	...	1010	...	600
17	...	—	...	—	...	—
18	...	28 "	...	1020	...	560
19	...	40 "	...	1016	...	640

Date.		Quantity.		Sp. gr.		Solids.
Oct. 20	...	52 oz.	...	1022	...	1144
21	...	68 "	...	1010	...	680
22	...	48 "	...	1014	...	672
23	...	40 "	...	1012	...	480
24	...	—	...	—	...	—
25	...	48 "	...	1012	...	576
26	...	40 "	...	1018	...	720
27	...	—	...	1012	...	—

CASE 53.—*Paralysis agitans; hypertrophied heart; high arterial pressure; normal urine.*—Eleanor F—, æt. 71. Dr. Wilks (Clin. clerk, Mr. Pizey). March 10th—April 20th, 1880. A washerwoman. Father died at 74, of "old age." Mother was subject to fits, and died in one, at the age of 70. One brother died of consumption, and a sister had a fit and died five weeks after it, having lost her speech and the use of the right side of the body. She has been in the hospital with erysipelas, but she forgets when. She had what she calls "rheumatism" some years ago, which has left some of the finger-joints in each hand enlarged (gout?). Has always been a very nervous woman. Has been temperate. She is a rather short old woman, with white hair and eyebrows and dark eyes. She has paralysis agitans affecting the right arm only. The apex of her heart is much displaced outwards, the second sound is accentuated. The pulse is very hard and persistent, the artery very thick. Urine pale in colour, sp. gr. 1020, acid, no albumen. On March 18th, 38 oz. were saved, sp. gr. 1020, no albumen. She had a prolapsus uteri, and with it some difficulty in holding her water; it was therefore found impossible to save and measure it. Further notes of the urine are made on March 19th and 23rd; on both occasions it was normal.

CASE 54. *Alcoholism; vertigo; high arterial pressure; albuminuria foretold and transient.*—Robert G—, æt. 40. Dr. Moxon (Clin. clerk, Mr. Bothamley). January 22nd—March 8th, 1879. A lighterman. Father and mother said to be rheumatic; latter died of heart disease. Patient had rheumatic fever two years ago; frequently has pain in the joints. Syphilis with secondaries seventeen years ago. Was

in Philip Ward with pleurisy a year ago. Has been in the habit of taking seven or eight pints of beer each day, besides spirits occasionally. He had a sudden attack of vertigo fifteen days ago, with some sickness at the onset; the giddiness has remained ever since and his gait has been staggering. He is a dark, healthy-looking, well-nourished man, with a somewhat dull expression. Lower eyelid rather puffy. His appetite is bad, and he is sometimes sick in the morning and always has nausea. He still has a rather uncertain staggering gait and complains of giddiness. There is no further note of the condition of his nervous system, but he was considered by Dr. Moxon to be suffering from alcoholism, and he gave no evidence of organic disease. "*Ophthalmoscopic examination.*—No retinal hæmorrhages. Discs normal." His apex beat is in fifth interspace, internal to nipple. There is no thrill or bruit. I made a note that "his pulse was extremely hard, persistent, and long; his second sound is accentuated intensely; his arterial tension is extremely high."



Pressure 3 oz.

Respiratory system normal. Urine pale in colour, clear acid reaction, sp. gr. 1015, no albumen, sugar, or blood. The arterial tension in this case was so high that I remarked to the clerks that in such a condition it probably only required a little exposure to cold to cause albumen to appear in the urine, a prophecy which was shortly verified. His urine was examined almost daily, and found free from albumen. A special note to this effect occurs on January 28th, 29th, 30th, February 1st, 2nd, 3rd, and 4th. On February 5th the following note occurs:—"A very slight trace of albumen is present in the urine. He was out in the park yesterday." The weather was raw and cold just then. On February 6th and 7th there was still a little albumen. February 8th.—The albumen has completely disappeared again. It was never found again during his stay in hospital, though constantly

watched for. He went out on March 7th. He has not the least unsteadiness of gait now and very seldom feels giddy.

CASE 55. *Dyspepsia ; hypertrophied heart ; high arterial pressure ; once a trace of albumen.*—Elizabeth I—, æt. 57. Dr. Wilks (Clin. clerk, Mr. Jackson). July 15th—August 9th, 1880. Has had several severe illnesses, the nature of which seems obscure ; she is said to have had rheumatic fever three times. She comes in for swelling of the abdomen and flatulence. She says that her feet have been swollen, and that her face is occasionally puffy ; they were not so at time of admission. Heart's apex displaced much outwards, impulse forcible and heaving in sixth interspace, about one inch or more external to nipple line ; second sound in aortic area very intense. No bruit. Pulse very persistent, long, and pushing. Artery thickened. Urine sp. gr. 1020, no albumen. July 21st.—Complains of pain in loins. Urine very pale in colour, sp. gr. 1010, a trace of albumen. July 31st.—Urine sp. gr. 1010, very pale straw colour, no albumen, slight quantity of phosphates. The report fails to give anything like a complete record of the urine. I believe it was tested more frequently and found free from albumen on all occasions.

CASE 56. *Severe recurrent attacks of vomiting and pyrosis ; high arterial pressure ; temporary aortic regurgitation or pericarditis ; urine normal.*—Mary P—, æt. 42. Dr. Pavy (Clin. clerk, Mr. E. Starling). April 16th, 1879. At intervals till May 2nd, 1880. She had good health till three or four years ago, when she was attacked with nausea and vomiting of a clear fluid, sometimes acrid, at others tasteless ; sometimes she would bring up very little, at others as much as half a pint. These attacks would sometimes last for several weeks and then leave her. During the attacks even the sight of food would cause nausea. Says she has lived principally on bread and cocoa. On admission she was quite unable to take food, frequently vomiting a clear fluid of alkaline reaction containing abundance of *Torula cerevisiæ* ; has a cold, numb sensation at the epigastrium, and pain up the œsophagus. The precordial dulness extended from fourth space to apex in sixth space, and

from middle line of sternum to just internal to nipple. Apex beat can be felt but not seen in sixth space, about one inch internal to nipple. First sound clacking, second sound loud and accentuated. Pulse very long and pushing, artery thickened. The sphygmograph showed the pulse to be one of exceedingly high pressure, the cardiac systole being much prolonged.



Taken with Pond's sphygmograph. Pressure not recorded.

There was subconjunctival œdema but none elsewhere. Urine of normal colour, sp. gr. 1024, no albumen, sugar or bile, contains indican, a mucous cloud, no casts. She seems to clip her words somewhat, there is slight external strabismus of right eye. She continued constantly vomiting clear fluid and suffering much from heartburn, taking nothing but a little milk and lime water. April 17th.—Last night vomited a little clear alkaline fluid with abundance of torula. April 19th.—Last night vomited about a pint of dark greenish coloured viscid fluid of acid reaction, showing under the microscope fat globules and torula. April 21st.—Dr. Pavy called attention to the frequent sighing and the external strabismus, with the nausea and vomiting on an empty stomach. He says that there may be some cerebral mischief. April 24th.—Patient says that she felt as if something burst in her throat last night; she then felt better. April 25th.—Is much better to-day, she feels as if she could eat an egg, hitherto having only taken milk and Liq. Calcis. May 5th.—Has had meat for four days and has had no nausea or vomiting since April 25th; feels and looks much better. May 11th.—Left, apparently quite well. Readmitted on Oct. 15th, 1879. Her illness was merely a repetition of that which she suffered in April. Her pulse was still one of high pressure. On examining the heart a to-and-fro bruit was heard down the sternum and most distinct a little to the left of the lower part of this bone. There is no note of any displacement of the impulse of the heart, and I cannot remember that any was found. October 18th.—The vomiting still persists. There is a little ptosis of

left lid. Optic discs examined and found to be normal. On October 23rd the vomiting had ceased. On November 2nd the vomiting again returned; on the 8th she had diarrhœa. Nov. 10th.—Diarrhœa has continued and patient looks very weak. Vomiting has increased very much. She states that the presence of food in the stomach does not cause pain and that the vomiting is generally worse when the food is absent. November 15th.—She is a little better and does not vomit as much as she did. Vomiting is not excited or increased after food. November 29th.—She was discharged to-day, having steadily improved. There has been no return of the vomiting. The bruit can be heard but is very indistinct. Her urine was carefully watched during her stay in the hospital. The observations are recorded in the following table:

Date.	Quantity.	Sp. gr.	Solids.	Alb.
Oct. 29	... 38 oz. ...	1016	608	None.
30	... 43 " ...	1012	516	"
31	... 40 " ...	1020	800	"
Nov. 2	... 38 " ...	—	—	"
4	... 40 " ...	1025	1000	"
5	... 34 " ...	1018	612	"
6	... 34 " ...	1018	612	"
7	... 28 " ...	1025	700	"
8	... 20 " ...	1030	600	"
9	... 28 " ...	—	—	"
10	... 20 " ...	1034	680	"
11	... 26 " ...	1035	910	"
13	... 13 " ...	1028	364	"
14	... 11 " ...	1028	308	"
16	... 16 " ...	—	—	"
17	... 16 " ...	1027	432	"
18	... 24 " ...	1022	528	"
19	... 14 " ...	1028	392	"
20	... 26 " ...	1023	598	"
21	... 34 " ...	1022	748	"
22	... 40 " ...	1020	800	"
23	... 46 " ...	—	—	"
24	... 38 " ...	1024	912	"
25	... 33 " ...	1025	825	"
26	... 51 " ...	1020	1020	"

Readmitted March 20th, 1880.—Her symptoms were the same as before, the attack was not quite so severe. On admission I have noted that the to-and-fro aortic bruit pre

viously audible could not now be detected. I never heard it during her stay on this occasion. Her pulse was still one of high pressure and the second sound was accentuated. It was noted at the time of her admission on this occasion that her urine contained *albumen*, amorphous urates, and phosphates. I doubt whether much reliance can be placed on this observation; the report is a very imperfect one, no further note of albumen is made, and it is said to be absent at the time of her discharge. I think that a cloud of phosphates produced by heat was mistaken for albumen. She was discharged well on May 2nd, 1880.

CASE 57. *Strangulated hernia; operation; great hypertrophy of heart; thickening of arteries; fairly good kidneys; death.*—Francis H—, æt. 60. Mr. Howse (Clin. clerk, Mr. Richardson). March 16th; died March 18th, 1880. He was admitted for hernia, which had been down thirty-six hours; an operation was performed on the night of admission, and he died two days afterwards. His son says that he had always had good health; he never remembers him ill before.

Post mortem (by Dr. Hilton Fagge).—After describing the local conditions of the bowel and peritoneum, the report continues: "The heart was much hypertrophied, weighing 18½ oz., the left ventricle being extremely thick, but also the left auricle. Valves all healthy. The kidneys appeared healthy, but the arteries in them were thickened. The microscope showed that there was some degeneration of certain Malpighian tufts, and perhaps a little excess of stroma in the kidney, but their structure was practically healthy. The arterioles of the pia mater showed moderate thickening, chiefly of the muscular coat. I think the case is distinctly opposed to the view that cardiac hypertrophy is secondary to an advanced degree of kidney disease."

VIII. CASES OF TYPICAL CHRONIC BRIGHT'S DISEASE, WITH WELL-MARKED ALBUMINURIA, WHICH WAS VARIABLE OR DISAPPEARED WHILE UNDER TREATMENT.

CASE 58. *The third attack of dropsy and albuminuria, both of which disappeared under treatment; high arterial pressure; hypertrophy of heart? urine became normal.*—Bridget K—,

æt. 42. Dr. Hilton Fagge (Clin. clerk, Mr. L. E. Stephens). Nov. 27th, 1880—Feb. 23rd, 1881. She had worked for many years as a cleaner in the hospital. Admitted for general weakness and dropsy. Father died æt. 42 of consumption, also mother, æt. 52. Has had seven brothers and sisters, of whom three died of consumption. Patient had general dropsy when 15 years of age, again during a pregnancy when 28 years old, not again till present attack, which commenced in the summer of this year. For about six weeks she has perspired much at night, her night dress and hair being quite wet through in the morning. She has been out of sorts all the summer, complaining chiefly of "swimming in the head," puffiness under eyes in the morning, also of the hands and feet. Thinks that lately she has passed less urine than usual. She is a well-nourished woman, with a care-worn anxious expression, is sitting propped up by pillows, as she says her back feels very sore when she lies down. No œdema of feet, legs, or hands, slight puffiness of lower eyelids. There is considerable œdema in the lumbar region; if she gets out of bed and sits for a quarter of an hour, her feet begin to swell and get stiff, which swelling almost as quickly subsides on her return to bed. Cardiac impulse regular and strong, apex beat in 5th interspace, two inches from middle of sternum, in nipple line. Area of cardiac dulness not increased. There is accentuation of second sound and long first sound at apex. No murmurs. Pulse 56, regular, long, full, and compressible. Tracing shows exceedingly high arterial pressure.



Pressure 3 oz.; takes more.

Tongue flabby and tremulous. Appetite bad, cannot eat anything in the morning; she has not been able to do so for two or three years. Bowels confined for four days. Lung signs normal. She has had almost constant right-sided headache for the last three months. The urine when admitted contained about "half albumen." For several days she continued much in the same condition. Dec. 15th.—She feels better and has lost the severe headache; she sat up for an

hour last night; this morning there is œdema of the feet and legs; the abdomen is distended by fluid and measures 36 inches in circumference at the level of the umbilicus. On Dec. 18th she was ordered Tr. Ferri Perchlor. mxxv , Sp. Chlorof. mxx , Inf. Digitalis ʒj , Aq. ʒj , ter die and under this treatment, aided by Pulv. Jalapæ Co. ʒij at night occasionally, she rapidly improved, the quantity of urine increasing, and the amount of albumen steadily decreasing, till it shortly afterwards disappeared entirely. On Dec. 17th the urine contained one-third albumen, by the 22nd it was reduced to "a trace," and on the 30th there was none. On the 23rd the circumference of the abdomen had fallen to $30\frac{1}{2}$, and by Jan. 13th it had reached and remained permanently at $28\frac{1}{2}$ inches. She went out on Feb. 23rd, her urine having afforded for considerably over two months not the least indication of renal disease; this could only have been suspected from the persistent high pressure, the slight displacement of the apex beat, and the history.

Date.	Quantity.	Sp. gr.	Solids.	Alb.	Bowels.
Dec. 1	25 oz.	1026	650	$\frac{1}{2}$...
3	—	1028	—	$\frac{1}{2}$...
4	—	1026	—	$\frac{1}{2}$...
5	30 "	1028	840	$\frac{1}{2}$...
6	18 "	1028	456	$\frac{1}{2}$...
7	16 "	1024	384	$\frac{1}{2}$...
8	18 "	1028	504	$\frac{1}{2}$...
9	19 "	1028	532	$\frac{1}{2}$...
10	20 "	1026	—	$\frac{1}{2}$...
11	16 "	1024	—	$\frac{1}{2}$...
12	18 "	—	—	—	...
13	30 "	1020	600	$\frac{1}{2}$...
14	26 "	1024	624	$\frac{1}{2}$...
15	30 "	1024	720	$\frac{1}{2}$...
16	30 "	1026	780	$\frac{1}{2}$...
17	50 "	1018	900	$\frac{1}{2}$...
18	49 "	1012	588	$\frac{1}{2}$...
19	76 "	—	—
20	48 "	1014	672	$\frac{1}{2}$...
21	67 "	1014	938	$\frac{1}{2}$...
22	106 "	1012	1272	Trace	...
23	85 "	1012	1020	None	...
24	74 "	1010	740	"	...
25	64 "	1012	768	"	...

Date.	Quantity.	Sp. gr.	Solids.	Alb.	Bowels.
Dec. 26	87 oz.	1010	870	None	...
27	60 "	1012	720	"	...
28	52 "	1014	728	"	...
29	70 "	1014	980	"	...
30	52 "	1018	936	"	...
31	68 "	1015	1020	"	...
Jan. 1	33 "	1020	660	"	0
2	33 "	1020	660	"	1
3	35 "	1016	560	"	3
4	42 "	1018	756	"	1
5	51 "	1012	612	Trace	0
6	62 "	1015	930	None	1
7	65 "	1012	780	"	0
8	60 "	1018	1080	Trace	0
9	50 "	—	—	None	0
10	57 "	1015	855	"	0
11	35 "	1018	630	"	2
12	44 "	1016	704	"	1
13	43 "	1016	688	"	2
14	43 "	1018	774	"	1
15	57 "	1015	855	"	0
16	56 "	1016	896	"	1
17	49 "	1018	882	"	0
18	40 "	1018	720	"	1
19	50 "	1012	600	"	1
20	49 "	1012	588	"	0
21	48 "	1012	576	"	1
22	40 "	1004	560	"	1
23	34 "	1012	448	"	6
24	45 "	1012	540	"	0
25	44 "	1012	528	"	0
26	44 "	1012	528	"	1
27	68 "	1014	952	"	0
28	72 "	1014	1008	"	0
29	36 "	1014	504	"	1
30	48 "	1012	576	"	0
31	44 "	1012	528	"	0
Feb. 1	32 "	1014	448	"	1
2	52 "	1012	624	"	0
3	52 "	1012	624	"	1
4	48 "	1016	768	"	0
5	36 "	1012	432	"	3
6	52 "	1012	624	"	0
7	56 "	1012	672	"	0
8	64 "	1014	896	"	0
9	60 "	1012	720	"	0
10	48 "	1012	576	"	0

Date.		Quantity.		Sp. gr.		Solids.		Alb.		Bowels.
Feb. 11	...	60 oz.	...	1012	...	720	...	None	...	2
12	...	40 "	...	1012	...	480	...	"	...	1
13	...	48 "	...	1012	...	576	...	"	...	0
14	...	40 "	...	1014	...	560	...	"	...	1
15	...	40 "	...	1012	...	480	...	"	...	1
16	...	36 "	...	1012	...	432	...	"	...	0
17	...	46 "	...	1010	...	460	...	"	...	1
18	...	60 "	...	1012	...	720	...	"	...	1
19	...	40 "	...	1012	...	480	...	"	...	1
20	...	56 "	...	1012	...	672	...	"	...	0
21	...	42 "	...	1012	...	504	...	"	...	1
22	...	44 "	...	1012	...	528	...	"	...	1

CASE 59. *Typical Bright's disease; hypertrophied heart; retinal hæmorrhages; albuminuria; temporary disappearance of albumen.*—Sophia Sanger, æt. 57. Dr. Pye-Smith (Clin. clerk, Mr. Jackson). August 11th—September 21st, 1880. Her father was subject to rheumatic gout. She has always had good health until about a year ago, when she began to lose her sight—she found she was unable to thread her needle. She applied to an ophthalmic hospital, where she was told that glasses would be of no use to her. She is unable to distinguish the face of any one standing close to her. Passes a good deal of urine in small quantities and frequently. Complains of pains in the back and loins, and of nausea in the morning. She is a thin, anæmic woman. There is a little œdema about the left ankle, none elsewhere. Apex beat in 6th space, about one inch external to nipple. The heart is not regular, missing a beat now and then. There is a systolic bruit heard at apex and in axilla, but not in the back. There is also a systolic bruit audible at base and in the neck. The second sound is accentuated. Pulse persistent and incompressible, the artery very much thickened and arterial pressure extremely high. Lungs said to be normal. Passes a fairly large quantity of pale straw-coloured urine, opaque and cloudy, sp. gr. 1009. Albumen present in considerable quantity. The eyes were examined by Mr. Higgins on August 22nd. Right eye, glistening white patches and spots in retina, especially about yellow spot. One rather large branch of vessel in right eye looks like a white cord; it appears obliterated or surrounded by an opaque material. On August 13th, 14th,

and 16th albumen was present in the urine, but in decreasing amount, sp. gr. ranging from 1007 to 1015. On August 17th, 18th, 19th, no albumen could be detected in the urine, sp. gr. each day 1010. August 21st.—A very small amount of albumen present. August 23rd, 24th and 25th.—No albumen could be found. After the last date it reappeared, and continued present in large quantity (about $\frac{1}{2}$) until she was discharged. The sp. gr. varied between the limits mentioned above.

CASE 60. *Acute attack of Bright's disease at 60; dilated heart; mitral regurgitation; albuminuria, which completely disappeared.*—Charles B—, æt. 60. Dr. Pavy (Clin. clerk, Mr. David). June 10th—October 6th, 1879. A coachman and gardener. Family history unknown. Last September (nine months ago) he caught "a severe cold," and was laid up in bed till Christmas time. He has been up once or twice since for a short time, but always relapsed; recently his cough has been very bad, and his sputa streaked with blood. He sits up in bed looking distressed and breathing with difficulty; his conjunctivæ are slightly jaundiced and œdematous. Cheeks have a bright colour from dilated venules. Fingers clubbed and nails curved. Impaired expansion of chest; general signs of bronchitis, with dulness at right base behind, and moist râles most abundant in this area. Cardiac dulness is said to be normal; the impulse of the heart cannot be felt. Heart sounds very faint, irregular rhythm, occasional systolic bruit. Pulse 60, irregular, hard, and incompressible. Urine slightly high-coloured, sp. gr. 1018, albumen abundant. He continued in much the same state till June 30th, when it is noted that his urine contained "no albumen." July 1st.—He has passed four pints of urine in last twenty-four hours. On the 3rd albumen has again appeared. July 9th.—Heart's action very irregular, tumultuous, and rapid. July 11th.—There is dulness over the bases of both lungs, and moist sounds are audible here. July 19th.—It is noted that a loud bruit is heard at the apex. July 31st.—Patient looks very ill. Conjunctivæ yellowish; breathing troublesome; abdomen swollen, and appears to contain fluid; liver enlarged. Passes only about three-quarters of a pint of urine

daily, high coloured, sp. gr. 1026, albuminous. Bruit heard as before. August 8th.—Patient continues very ill. Urine getting rather more abundant, a pint and a half in twenty-four hours, sp. gr. 1020. Moist sounds over both bases, extending to within two inches of spines of scapulæ. Bruit not so loud. About the beginning of September the patient commenced to improve. On September 12th he is feeling much stronger. Albumen very much less, only about one-sixth. He gets up now of an afternoon. September 14th.—Still improving, he gets up every day. No albumen in urine, but some deposit of urates. September 26th, 29th, and October 5th.—Each day he is said to be improving, and his urine is found to be normal. He went out on the latter day, his breathing much relieved.

CASE 61. *Acute renal dropsy, probably chronic disease; blood and albumen variably present, their presence determined by leaving the bed.*—Charles V—, æt. 44. Dr. Habershon (Clin. clerk, Mr. S. Thomas). February 10th—April 7th, 1879. A labourer. Has been a great drinker, chiefly of spirits. Family history unknown. He has had smallpox, famine fever, and ague. Was in Guy's under Dr. Owen Rees eleven years ago with pleuro-pneumonia. He suffered from stricture of the urethra fourteen years ago. He has been subject to cough every winter for the last eleven years. He has been in the habit of weighing himself daily, his ordinary weight varying between 9 st. 12 lbs. and 10 st.; three weeks ago he was astonished to find himself 10 st. 9 lbs.; he had been getting heavier for four days. Two days after this he noticed that his face and the back of his hands were enormously swollen, and that when washing himself his abdomen shook about; he also passed very little urine, and what he passed was of very high colour. He came up to the out-patients', and after a week's treatment was recommended for admission. He is a pale, fairly nourished man, his face is puffy, and his feet and legs cedematous. Liver one inch below ribs. Abdomen flaccid. Chest barrel-shaped, hyper-resonant, especially at left apex, where there are some moist sounds audible. Area of cardiac dulness rather increased. Apex beat detected with difficulty, it appears to be in 5th

space and a little internal to mammary line. Second sound accentuated. Pulse very persistent and cord-like, long and incompressible. Urine small in quantity, pale straw-colour, sp. gr. 1012, contains a small quantity of albumen. In four days the œdema had completely disappeared from the legs. The patient steadily improved. On March 7th the albumen had completely disappeared from the urine. It remained absent on March 11th. On the 17th a trace of albumen, and also of blood, had appeared in the urine, sp. gr. 1025. On the 24th and 25th the urine contained no albumen, but gave a slight reaction with the guaiacum test. Again a trace on the 26th, much on the 31st, but neither albumen nor blood on April 4th. It had reappeared again on April 7th, when he was discharged. This variation is no more than is commonly seen in the albuminuria following scarlatina, and in this case it was determined, as it often is in the scarlatinal cases, by leaving the bed; he was free while he stayed in bed, whereas he had albuminuria on getting up and going out. In scarlatinal convalescents I have repeatedly watched this change from normal to albuminous urine; half an hour out of bed is often long enough to produce it. The rapidity of this change is too great for the kidney condition determining it to be a structural one; it must be functional, or more probably a vascular phenomenon. The fact stands, therefore, that *the same kidney, and that a diseased one, can produce alike normal and albuminous urine.*

DESCRIPTION OF PLATES.

(From drawings by Mr. GEORGE TURNER.)

These Plates are intended to show different degrees of the vascular and interstitial changes which are produced in kidneys subjected to the influence of prolonged high arterial pressure, *without nephritis*.

Plate I shows the changes in a well marked degree; Plate II shows the same changes in a much earlier stage.

The changes present in my specimens are as follows:—The *membrana propria* of the tubules is thickened both in the cortex and in the medulla; there is also inter-tubular fibro-hyaline thickening.

There is a similar hyaline thickening of the *intima* of the arteries, sometimes of the *adventitia*. There is thickening of the muscular coat of the smaller arterioles.

There is fibro-hyaline thickening of the capsules of the Malpighian tufts, involving the vessels of the *glomeruli*, and in some cases obliterating them.

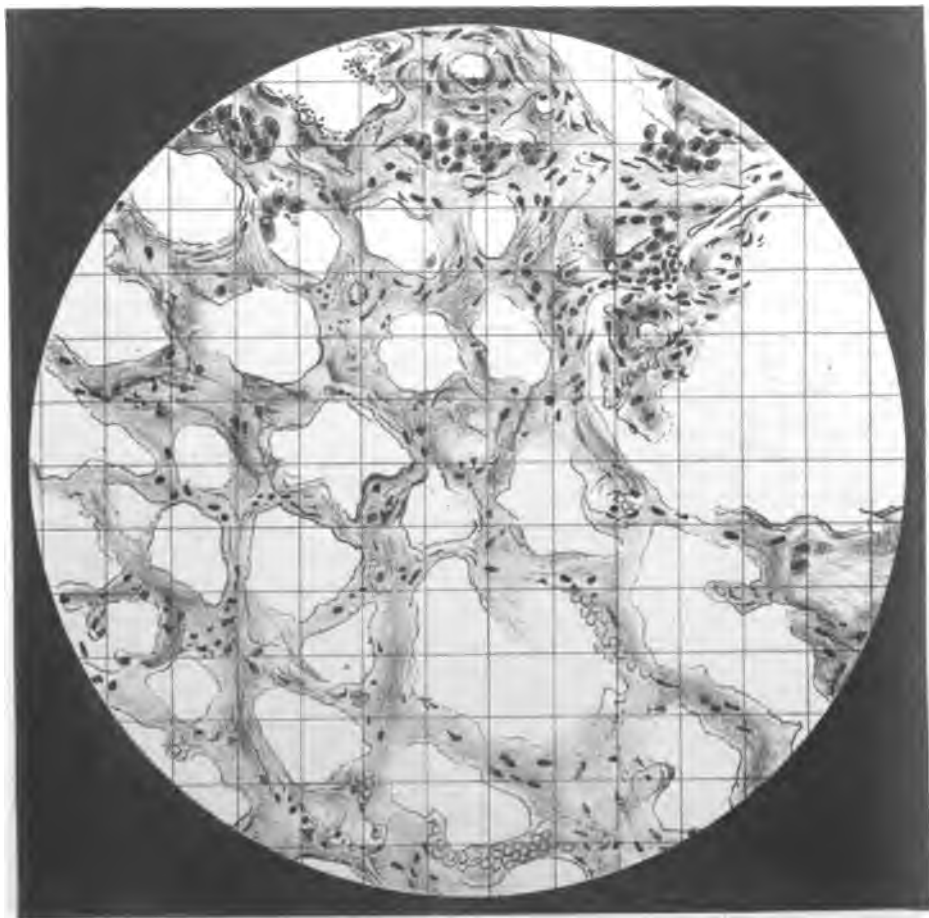
There is very little, if any, small-celled growth visible in the specimens.

These changes are not all shown in the Plates.

fig.

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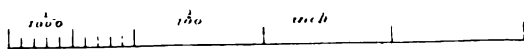
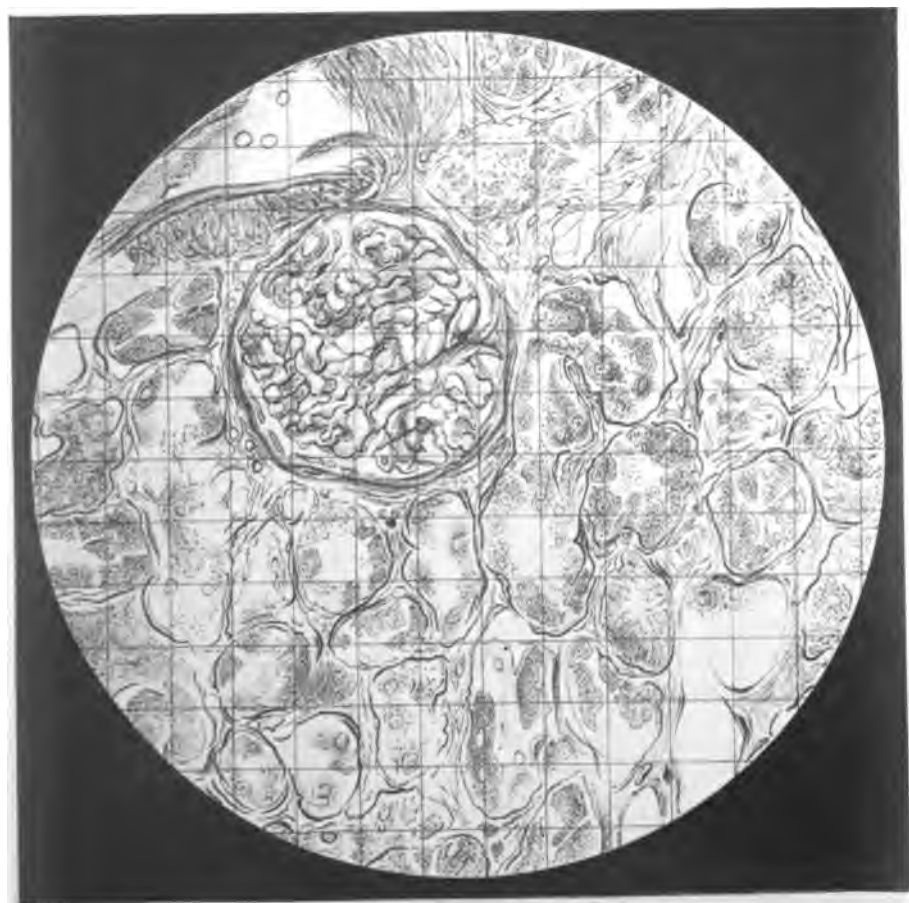
PLATE I



100µ 1mm

Case 1.

PLATE II



Case 49

4

ON A REMARKABLE INSTANCE
OF
HEREDITARY TENDENCY
TO THE
PRODUCTION OF SUPERNUMERARY DIGITS.

By R. CLEMENT LUCAS, B.S.

THE irresistible tendency of the offspring to resemble their parents is so well recognised that it is a divarication from the type rather than a resemblance which is apt to excite comment. The height, the build, the features, the complexion, the constitution, are all traceable to one or other parent, as well as such minor details as the size and direction of the teeth, the shape of the nails, the growth of hair and the time at which it may decline.

Certain mental attributes are scarcely less obviously hereditary. Galton has collected many interesting facts in support of the belief that genius is hereditary, and specialists in mental diseases are not lacking in proof that lunacy is transmitted from generation to generation. The hereditary tendency to certain diseases is fully recognised, but the recurrence of deformities in successive generations has, perhaps, excited less interest and received less attention than it deserves. Among minor deformities I have more than once traced a crooked little finger through three generations. It is seldom that one meets with so intelligent a parent and one so well acquainted

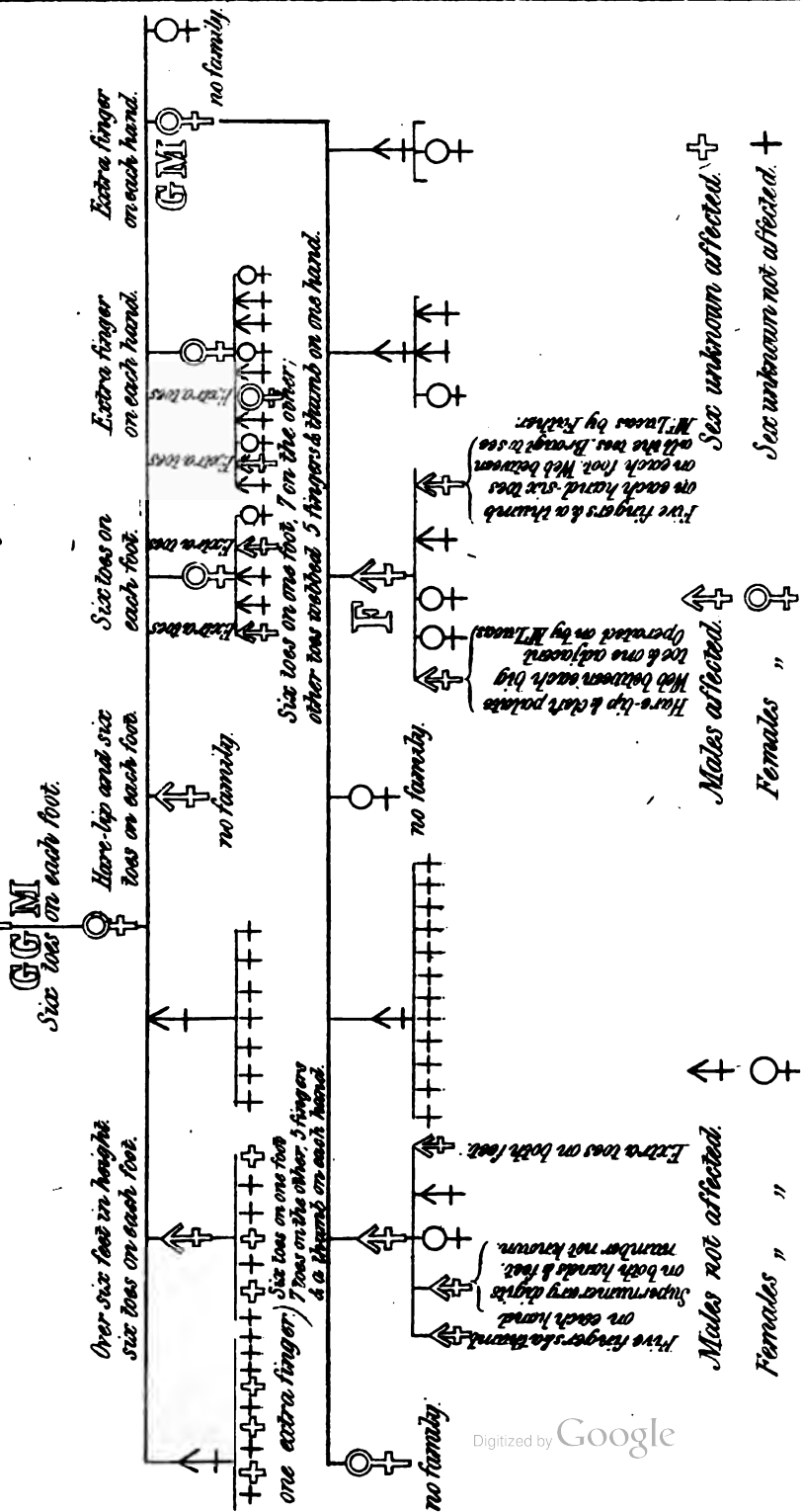
with his family history as the man who furnished the accompanying genealogical table. For the details of the history and the strikingly clear manner in which the pedigree is shown, I am indebted to my dressers, Mr. Key and Mr. Richardson. The liability to the production of supernumerary digits was well known in the family to which this man belonged, and when an infant was added to his family his first thought was to examine its feet and hands.

I am made aware through the prospectus of lectures given at the Royal College of Surgeons that Mr. Hutchinson delivered this spring lectures on heredity in which attention is drawn to this abnormal development, but I was prevented from attending these lectures, and as they have not yet been published I am unable to allude to the results of his observations.

It will be seen on referring to the chart that supernumerary fingers and toes can be traced in this family through five generations. It was transmitted through the maternal great grandmother, the grandmother, and mother to the man who furnished the history, and whose children were brought under my observation. Altogether the great grandmother of my patients appears to be responsible for abnormalities occurring in no less than twenty-four persons out of a total of eighty descendants, or thirty per cent. of those carrying her blood. Considering the number of times the blood has been diluted by marriage, the persistency with which this tendency asserts itself is very remarkable. The maternal grandmother had a family of eight, five of whom were affected, viz., two sons and three daughters. Her eldest son, though himself normal, had three children affected out of a total of nine. The second son, who was over six feet in height and had six toes on each foot, had seven children, three of whom were affected. The third son escaped, and his family of seven also escaped. The fourth son, in addition to six toes on each foot, was the subject of harelip; he had no family. The eldest daughter had six toes on each foot. She had four sons and a daughter. Two sons, the first and the fourth, had extra toes, whilst the second and third sons and the daughter, who was last born, escaped. The second daughter had an extra finger on each hand. She had a family of ten, six sons and four daughters, of whom two only

GGG M GG.U.

† Said to have been a giant and exhibited.



were affected, the second a son, and the fifth a daughter. The third daughter was the grandmother of my patients; she was born next to the last and had an extra finger on each hand. The youngest daughter was unaffected and remained unmarried.

The grandmother gave birth to seven children, five sons and two daughters. The eldest, a daughter, had one extra finger, and she had no family. The second, a son, had six toes on one foot, seven on the other, and five fingers and a thumb on each hand. He had five children, four sons and a daughter. Three of the sons, the two elder and the youngest, were the subjects of supernumerary digits, whilst the daughter and one son escaped. The third, a son, was normal, and his twelve children were all normal also. The fourth was a female, who escaped; she had no family. The fifth was the father of my patients. He had six toes on one foot, seven on the other, with the inner toes webbed, five fingers and a thumb on one hand. His eldest son had harelip and cleft palate, and a web between the big toe and one adjacent in each foot. The second, third, and fourth children, two girls and a boy, escaped. The youngest son, an infant, was born with five fingers and a thumb on each hand, six toes on each foot, and a web between all the toes. The sixth son (uncle of those last described) was normal, and he had three normal children. The seventh and youngest son was also normal, and has one normal daughter.

It is worthy of note that the cleft palate and harelip present in one of the children described, was foreshadowed in a harelip, which occurred in a great uncle.

As it is probable that a tendency to the production of superfluous fingers and toes runs in but few families, and as other observers may meet with different branches of the same family I think it right to publish the names of those whose descendants have incurred a liability to this deformity.

The family came originally from Suffolk, and the maiden name of the great-great-grandmother mentioned in the chart was Dawley; after marriage her name was More. The grandmother's maiden name was Collins and the tendency shows itself in her brothers and sisters named Collins, Mendham, and Hammond. She married a Phillips and there are eight of this name in whom the deformity has shown itself.

ON CHRONIC NASAL OBSTRUCTION:

I. OZÆNA.

II. ADENOID GROWTHS OF THE NASO-PHARYNX.

By C. H. GOLDING-BIRD, B.A., M.B.

CHRONIC obstruction to the nasal passages is a very common clinical symptom, complained of by patients, of a variety of pathological conditions, which, as a rule, only careful investigation can differentiate; and their further frequent statement—without foundation—that they have polypus, is at first misleading.

A record having been kept of such cases during especially, though not exclusively, the last three years, the following analysis and classification may be made of them. It will include all those conditions that commonly are seen in ordinary hospital and out-patient practice. It therefore does not include very unusual states, *e. g.* the presence of rhinoliths.

“STUFFINESS” OR CHRONIC NASAL OBSTRUCTION.

1. Usually without any discharge and not variable with the state of the atmosphere.

Tumours (except gelatinous polypi), such as papillomata, enchondromata, sarcomata, nævi.

Displacements or bendings of septum nasi, natural or acquired.

Chronic inflammatory thickening of mucous membrane of inferior spongy bone.

2. With anterior nasal discharge of watery character, worse in damp weather.

Gelatinous polypi.

3. With posterior nasal muco-purulent discharge, especially of a morning; 'dead pronunciation.'

Adenoid growths of the naso-pharynx.

4. With fetid anterior nasal muco-purulent or sanguineous discharge.

The varieties of *ozæna*.

Only the two last-mentioned classes will in this communication be illustrated with cases and remarks.

OZÆNA.

Ozæna includes all cases of offensive rhinorrhœa. It is a clinical expression for complex pathological facts. It may affect one or both nostrils, and is, as will be shown, mostly constitutional in origin, although some accidental circumstance may be the immediate provoking cause.

Since the strumous diathesis is credited with a large share in the production of *ozæna*, an appreciation of this condition is first necessary. The scrofulous or strumous and the tuberculous were formerly, and are still by some, considered as different diatheses, but a more extended pathological knowledge, coupled with a less firm faith in complexions and physiognomies, is causing both to be considered as one; and without now going into the arguments *pro* and *con*, it may be mentioned that the histological likeness of tubercle to many of the local scrofulous manifestations, and the frequency of strumous children being born of tuberculous (phthisical) mothers, are of themselves facts of no slight importance.

For the purposes of this communication the word strumous will be used as expressing this one diathetic condition.

Like all diatheses—and as to the matter of that, like every-
in pathology—the strumous diathesis is but a breach in

the fulfilment of a fundamental physiological law, in this case the law of growth; and we may thus express it from its three points of view. *Physiologically*, it is an imperfect fulfilment of the law of development; *clinically*, it is known as a condition of bad repair and bad resistance, or, in other words, slowness in healing, with a tendency to very chronic inflammation on the slightest provocation; *pathologically*, it is a dyscrasia of the lymphatic system. Regarding the last, it must be said that the word "dyscrasia" is a convenient limbo to which to consign all those undetermined conditions that are awaiting elucidation, and commits the user of it to nothing more than that there is "something wrong." There are many manifestations of the diathesis which at once suggest the lymphatic system as the one at fault; in others it is not so evident, and this arises from the fact that they are in the form of inflammations and modes of repair, which one is so accustomed to associate with the blood-vascular system. It must suffice at present to say, however, that evidence is accumulating rapidly which shows that not the blood, but the lymphatic-vascular system, is the more important factor at work; and this admission once made there is no difficulty in associating a chronic strumous catarrh of a mucous membrane, with the more evidently lymphatic process of chronically enlarged glands, whether in the same or different individuals.

A liability to chronic catarrh of the mucous membranes is a prominent symptom or mark of the strumous condition; and considering how common in the population generally a "cold" is, and in how very few proportionately it assumes a downright chronic character, the custom has been—and a very proper and reasonable one too—to regard those cases that become chronic as belonging to the strumous diathesis, whether they show or not a particular cast of countenance, or shade of complexion, or have a particularly suitable family history. Now, in a very common form of ozæna, to be directly described, the patients almost always state that it began as a cold in the head; and hence some writers on the subject class these by themselves as showing the results of catarrh of the nose or coryza, and put the strumous cases separately, although there is, by the time they appear before the surgeon, no evident clinical difference between them.

It seems a pity that such a distinction should be made, and the more so as the reason given for cases of coryza appearing later on an ozæna is that they are strumous, while no practical advantage is gained by such a classification. If also the surgeon takes one patient's word that the disease began in a cold, and thereupon puts it down as catarrhal, and then another's that he had no cold and calls it strumous, while both at the time of observation give the same objective symptoms, it is manifest that a distinction is made upon the least reliable of all clinical grounds. The objective symptoms are usually sufficiently evident to enable each case to have its place allotted to it by these means; and where the same local changes may belong to two different remote causes, then the history of the patient, *e. g.* as to struma or syphilis, must settle the question. Such a method is adopted here.

While as at first stated all cases of offensive rhinorrhœa belong to the *genus* ozæna, only those in which the origin is intrinsically nasal will be here mentioned; and thus cases (such as necrosis of the hard palate) beginning elsewhere, but ending in the nose, will not be taken into account.

The patient suffering from ozæna generally complains of stuffiness in one or both nostrils, more frequently the former, a foul state of the breath, and a stinking muco-purulent or sanguineous discharge from the anterior nares, and only occasionally down the throat. Accompanying the discharge are "crusts" or scabs, or even pieces of bone. The "crusts" appear with tolerable regularity, being formed in a few hours in some cases or a day in others; they are blown down or removed by the patient, new ones then taking their place. In any one case the "crusts" are mostly of one size, as though formed always over the same—and that a limited—area. Bone when discharged is only so at long intervals, perhaps once or twice in many months, but the patients often call their "crusts" bone. They further, in by far the larger proportion of cases, volunteer the statement that they are suffering from polypus, but the absence of an anterior watery discharge alone will suffice to render the surgeon sceptical. The foul smell, so obvious to his friends, is at times denied by the patient; this is from one of two causes: there is anosmia either from inflammation having destroyed the function of the

Schneiderian membrane, or from the patient himself being so used to the odour that he no longer appreciates it; this last is known by testing him with other odoriferous substances. Where, however, real anosmia exists, the appreciation of flavour—taste, as the patient says—is also lost, the vapour of the substance tasted, though carried up through the posterior nares, fails to excite smell, thence the inability to recognise “bouquet” or “flavour;” taste, pure and simple, is always intact. Occasionally there is severe frontal “stuffiness” headache, worse on stooping. Tenderness of the nose is a very variable symptom; sometimes the nares are excoriated, either from the cause of the ozæna or the result of the acrid discharge; there may be acute external swelling and redness, or pain is complained of on pressing on the nose at various parts. One spot of pain, however, it is very important to recognise, since it points to an extension of the inflammatory process. It is tenderness on firm pressure over the frontal sinuses, at times, but not always, accompanied with similar tenderness on the bridge of the nose. It is not a mere superficial pain; there is nothing visible and no cutaneous redness, but firm pressure at once makes the patient call out, when before he probably was not aware of anything wrong so high up.

This indicates inflammation in the muco-periosteum of the frontal sinuses, and being a late symptom points to extension having taken place from the middle meatus up the infundibulum. In such cases it may be here anticipated that the surgeon is even more than ever guarded in his prognosis, which, omitting the frontal symptom, is already not too cheering.

There may be visible deformity of the nose, apart from the appearances of acute inflammation already mentioned. The usual chronic changes are sinking of the bridge of the nose, a general “spreading out” or thickening of the bridge and cartilaginous parts, resembling the appearance seen in polypus cases, or a lateral displacement of the central parts, sending the point of the nose quite to one side.

While the deformity may be the result of an actual necrotic process, as is usually seen in syphilitic cases, it may certainly occur without any evidence of this having taken place. The increased breadth of the nose, while following loss of the septum, may also result from thickening of the tissues cover-

ing the nasal bones; and the lateral bend in the septum appears to be due to the chronic muco-periostitis or perichondritis having so affected the cartilage that it loses its natural stiffness and resiliency. This is but surmise, there being no histological evidence of it as yet; but it is certainly untrue that all cases of displacement are caused by loss of tissue by necrosis. The history of the case, together with direct observation, can often disprove this.

The following cases are selected from about thirty consecutive ones, mostly occurring in my out-patient practice.

They are thus arranged :

I. *Constitutional ozæna.*

a. Strumous.—This manifests itself in the *catarrhal* or in the *impetiginous* form. Two of the impetiginous cases were immediately after scarlet fever, and might therefore claim a separate place as *exanthematous*.

b. Syphilitic.—This again shows two forms, the *catarrhal* and the *muco-periosteal* or *gummatous*.

II. *Local ozæna.*—This includes foul nasal discharge from blows or fractures, foreign bodies, as clots, plugs, sloughing tumours.

I. CONSTITUTIONAL OZÆNA.

a. Strumous.

The simplest case noted was that of a gentleman, about 19 years of age, whom I saw in 1876. Six weeks before, he had a cold, which in a month gave rise to an offensive rhinorrhœa from one nostril. Two days' constant inhalation of Vapor Iodi, however, cured him. It is exceptional to see a case so early, and certainly to cure it so quickly.

CASE 1.—Emily W—, æt. 21, out-patient, 16th May, 1881. Four years' history of phthisis. Delicate looking, but nothing markedly "tuberculous" in appearance. Two years ago had cold in head, followed by much nasal stuffiness and foul discharge, but confined to right nostril. Used to syringe with water and bring down "crusts." Breath offensive to

herself and others. Slight anosmia. Some pharyngeal discharge, which she hawks into the mouth.

Examination.—Right nostril: great congestion and puffiness of mucous membrane; inferior spongy bone swollen and granular; an abraded patch upon it, now covered with a scab. Left nostril: redness of mucous membrane of septum; scab on outer wall; inferior spongy bone normal. Digital examination of naso-pharynx showed nothing wrong except much sticky discharge on wall of pharynx.

Remarks.—A common type of ozæna. Though the abrasion mentioned had just the appearance of “granular lids,” yet at times ulceration is to be seen. Without a syphilitic taint I have never seen the ulceration descend deeper than the mucous membrane.

CASE 2.—Caroline S—, æt. 17, out-patient, 10th November, 1879. Crusts and foul breath for years (? five years), and gradual depression of nose just beyond nasal bones. Pain on pressure over bridge of nose. Partial anosmia. Last few weeks crusts have come down with the discharge on the right side only.

Examination.—Left nostril blocked by bulging of septum, so that outer and inner walls of nostril touch. Right side shows general velvet-like condition of all the visible mucous membrane, in part scabbed over; ? if ulcerated.

CASE 3.—Mary D—, æt. 14, out-patient, February 10th, 1879. A “strumous” looking girl; flat face, spreading nose, thick upper lip. No evidence of hereditary syphilis whatever. For years had pain in nose, and foul discharge and crusts.

Examination.—Marked pain on pressure over the frontal sinuses and bridge of nose. Anterior rhinoscopy showed nothing wrong.

CASE 4.—George H—, æt. 46, 14th July, 1879, out-patient. Cold in head last March; since then foul discharge from right nostril, with crusts. At times stuffy frontal headache, slight pain on frontal pressure.

Examination.—Anterior rhinoscopy showed nothing.

Remarks.—In the two last cases there can be no doubt that the same process was going on in the nose as in Cases 1 and 2, although, from the mere accident of position, it was impossible to see it. Both of them further give positive evidence of the high position of the disease by the frontal tenderness on pressure. Case 3 presented the cast of countenance, which when present is an accepted sign of the strumous diathesis, although its absence must not be taken as necessarily proving the contrary.

CASE 5.—Thomas L—, æt. 11, outpatient, 25th August, 1879. (Brother to Case 9). Usual ozæna symptoms after a cold six months ago. A heavy-featured, thick-lipped strumous-looking boy.

Examination.—Right nostril showed a congested mucous membrane and oblong ulcer about the size of a split pea on the septum; it was very tender to touch. Nothing seen on left side, though crusts come down both.

Remarks.—The disease here was bilateral, but only on one side could anything be seen. The ulcer showed a later catarrhal change than in the preceding cases quoted.

CASE 6.—Eliza C—, æt. 6, outpatient, 30th May, 1881. A fair-haired, pale, cachectic child. Has ulcerative stomatitis in lower jaw and simple stomatitis in upper jaw. Has had "breakings out" on face. Has had ozæna for many weeks; muco-sanguineous discharge, crusts.

Examination.—No external swelling; anterior rhinoscopy showed spongy state, with swelling of muco-periosteum of both sides of septum, and similar state of right inferior spongy bone. No ulceration; no crusts seen.

Remarks.—Occasionally the coincidence of stomatitis with ozæna is seen. May such cases be taken as throwing a light upon the general pathology of catarrhal strumous ozæna? It is likely, at least, that the muco-periostitis is the same both in mouth and nose.

CASE 7.—Miss C—, æt. 16, 30th April, 1881. Had been for four months under treatment for early spinal caries. Within the last three weeks has had impetiginous eruption on upper lip and about the nostrils, which gradually spread up

the nose, and was followed by foul discharge and crusts. The crusts rapidly re-form if removed.

Examination.—Both nostrils nearly blocked with impetiginous scabs, and breach of surface seen in the septum (probably traumatic from picking).

CASE 8.—Miss F—, æt. 21, 23rd June, 1881. For a long time has been under treatment for scrofulous glands of the neck. Last summer suffered from eruption about upper lip and nostril, it spread up the nose and then followed foul breath and discharge and crusts; it was cured with some ointment (Ung. Hyd. Nit. dilut.). This summer it is just recommencing.

Examination.—Impetiginous eruption just inside the left nostril; slight external redness, no ozæna as yet.

CASE 9.—Ellen L—, æt. 7, out-patient, 7th June, 1880; sister to Case 5. One month ago had a "sore nose," followed by foul smell and discharge. Now only impetigo of nostril extending into nose to be seen. Same cast of countenance as her brother.

CASE 10.—James J—, æt. 8, out-patient, 13th June, 1881. A delicate-looking child; still looking pale from scarlet fever one month ago. Has patch of impetigo on right brow, and others about lips and nostrils. Foul smell; discharge of crusts and blood from nose. Frontal pain on pressure; frontal headache.

Examination.—Whole nose much swollen and very tender; both nostrils quite blocked with crusts. Naso-pharynx contains fine adenoid growths. On June 20th, the crusts being removed, in the right nostril there was much muco-purulent discharge seen, in the left some crusts; in both great hyperæmia of the mucous membrane. The whole nose much less swollen. No foul smell now.

CASE 11.—Annie M—, æt. 13, out-patient, 5th October, 1880. Scarlet fever one year ago; since then has been troubled with eruption about nostrils, stuffiness in breathing, discharge and crusts, but no bad odour.

Examination.—Each nostril completely blocked with impe-

tinginous crusts, coming from column of nose and alæ. No foul breath.

Remarks.—These five cases illustrate well the condition of ozæna from an impetiginous eruption. In Cases 7, 8, and 9, there were other manifestations of the strumous diathesis, and impetigo is of itself a recognised diathetic rash. In Cases 10 and 11 the disease followed upon scarlet fever; judging from the exanthematous manifestations in the mouth, we would rather have expected a severe muco-periostitis; the rash of impetigo may, therefore, have been little else than a coincidence. That both were, however, the same is worthy of note. Case 11 showed all the signs (save the foul odour) that go to make a case of impetiginous ozæna; and, as the odour depends on the accident of putrefaction, and not upon any intrinsic pathological peculiarity, it is introduced here. Similarly catarrhal cases may be seen in which the smell is absent. Such a case is the following:

CASE 12.—Rose H—, æt. 21, out-patient, 18th September, 1880. Suffering for six months with rhinorrhœa and discharge of crusts, but no bad smell.

Examination.—Right nostril appears normal. Left nostril shows mucous membrane spongy, injected, and easily bleeding; much muco-purulent secretion.

b. Syphilitic ozæna.

Constitutional syphilis is, next in frequency to the strumous diathesis, a source of ozæna. One class of cases is not to be distinguished, usually, from the strumous catarrhal, the history alone, unless there are other specific marks on the person, being the guide as to origin. That such should be the case would be expected on reflecting how like to the strumous are so many of the constitutional syphilitic manifestations—the lymphatic system in both markedly suffering; hence “bad repair,” “bad resistance,” and tendencies to catarrh. The cases here quoted illustrate the two varieties of ozæna commonly seen in syphilis, omitting mucous tubercle, which causes “snuffles,” and rhinorrhœa, but not bad odour, in

infants. They are the *catarrhal* and the *muco-periosteal* or *gummatous*.

As an example of the catarrhal the following case is given :

CASE 13.—Mary W. æt. 31, out-patient, 26th May, 1879. A case long under treatment for constitutional syphilis. One year ago had a cold, followed by rhinorrhœa that became offensive; then stuffiness in nose, relieved by “easing down with a hair-pin” some crusts that used to block it up. Breadth of nose has been gradually increasing, but there has been no sinking of the bridge.

Examination.—Nose as far as seen lined with crusts; superficial ulceration, and some impetiginous scales about the nostrils; these sores were subsequent to the other symptoms, and apparently due to the irritation of the discharge.

CASE 14.—John W—, æt. 29, out-patient, 2nd May, 1881. Contracted syphilis three years ago; has had rash and constitutional symptoms. Three months ago had complete stoppage of the right nostril, which continues at present. No discharge from nose; not worse in wet weather. No foul odour.

Examination.—Externally the right nostril, at junction of bony and cartilaginous wall, presents a tender, red, semi-fluctuating swelling, which from inside is seen to grow from the septum, to extend across the nostril, and protrude the outer wall, as above. The mucous membrane is red and inflamed. The left nostril is normal.

CASE 15.—William H—, æt. 40, out-patient, 23rd February, 1880. An old syphilitic patient. The last year has had stoppage of the left nostril, but no pain. Six months ago something burst in the nostril, discharging a teaspoonful of matter, and ever since then has had crusts and foul discharge come down. No pain even now, and no external swelling and deformity. No frontal pressure, pain, nor headache. When the nose first became bad he had tender lumps (nodes) on the head and forehead.

Examination.—In left nostril a large scab seen on outer wall, which was removed, and then the mucous membrane was there

found superficially ulcerated. No proof of any necrosis of bone as yet. He rapidly improved with iodide of potassium.

CASE 16.—Robert B—, æt. 30, out-patient, 13th June, 1881. Had some venereal complaint nine years since, but is very reticent on the point; can get no positive evidence of syphilis (unless in the nose) and the remedies. Has had pain in right nostril and right side of bridge of nose two months ago; at first it was all internal, but afterwards the outside of the nose became tender. Lately has had sensation of something running down throat, and last week had blood come from the right nostril, but no discharge of matter; very foul breath.

Examination.—Right nostril shows mucous membrane very injected; high up on septum is a conical truncated swelling of the muco-periosteum; and crater-like, at the top, is a depressed ulcer. It has the appearance of something having formed under the mucous membrane and subsequently burst outwards. In two weeks all his symptoms vanished with anti-syphilitic remedies, but the swelling had not quite subsided.

CASE 17.—Abraham S—, æt. 45, out-patient, 6th December, 1880. Syphilis seven years ago. For the last six years has had constant buzzing (like machinery in motion) in left side of head, better when in the open air than in a close room; lately this noise has extended over to the right side. Undiminished by pressure on carotid; no exophthalmos; no cranial bruit. The noise (pain, he calls it also) varies from day to day. When it began there was also very foul discharge from the nose; the bridge of the nose has been gradually sinking. Patient has pained expression; is very anæmic. He rapidly improved under iodide of potassium, and on April 25th, 1881, a piece of bone came away from nose. The septum is now perforated, and there is more bone loose.

Remarks.—These four cases give the stages of gumma in the nose ending in veritable ozæna. The absence of foul smell in Case 14 does not prevent its being quoted here as an early state of the disease. The other cases speak for themselves. Case 15 is regarded as one of gummatous periosteal abscess, suddenly discharging.

II. LOCAL OZÆNA.

Blows on the nose, as might be anticipated, account for some cases of ozæna. At times the injury at once produces death of tissue, and symptoms of ozæna: in other cases it gives rise to a muco-periostitis, resembling the ordinary catarrhal affection; perhaps in these last there is a strumous tendency or diathetic condition present. Thus—

CASE 18.—Francis S—, æt. 9, out-patient, 27th June, 1881. A year ago was struck on nose with the handle of a door; there was much epistaxis; since then the bridge of the nose has gradually been widening out. Three weeks after the accident offensive rhinorrhœa, with crusts and blood, commenced and still continues.

Examination.—No external redness, only the deformity of nose; internally only the usual catarrhal appearances seen; no proof of dead bone; the mucous membrane of both the inferior spongy bones was very thickened, so as on the left side to block the meatus.

In the following case ozæna followed the blow directly.

CASE 19.—Helen M—, æt. 25, out-patient, 19th May, 1879. A healthy woman; married; not syphilitic. Blow on nose six months ago; it did not bleed. Three weeks afterwards there was discharge of blood-clots and matter, and of crusts, with foul odour. The nose was then very painful and sore, and it has gradually altered in shape, the bridge sinking down. Six weeks later first felt loose bone in nostril.

Examination.—Pain on pressure over nasal bones. With the speculum the anterior inferior part of vertical plate of ethmoid is seen dead and exposed, but still fixed.

Other local causes for ozæna, that may be called accidental, need but be mentioned, as foreign bodies in nose and rhinoliths, retained blood-clot or sponges, shreds of mucous membrane sloughing after manipulation, and polypi imperfectly removed (or that have been tied) becoming gangrenous.

Treatment.—The mechanical difficulties met with, and not anything intrinsically hard to deal with in the pathological conditions of ozæna, are the causes of disappointment in the treatment of this disease.

Amongst all classes of life the same anatomically devious paths along which medicaments have to travel in the nose are found of course; but among the poor the want of time to give sufficient attention to treatment is a great drawback to success. Many, too, of this last class, upon losing the foul smell and knowing how to keep up the improvement, cease to attend the hospital, and so still further reduce the number of radical improvements and cures.

In all cases, cleanliness, even without antiseptics, is essential, and suffices to get rid of the smell. The nasal douche with glass rose ("watering-pot") nozzle is best; but the new form of Higginson's vaginal syringe, with a "rose" nozzle, answers well. A syringe with a single jet is all but useless, the fluid being sent along the inferior meatus and not thrown into the upper nasal passages.

After using whatever is ordered for cleansing the passages and removing the crusts, the remedies, if liquid, may be thrown up in the same way, or more efficiently as a spray.

For cleansing, weak salt and water is as good as anything, used in quantity; or carbolic lotion or Condy's fluid can be used instead; but these, whilst antiseptic, do not wash the mucous membrane so well as the weak alkaline solution of salt.

In catarrhal cases astringent lotions, as of alum and zinc, may be used in bulk after the nose is cleansed; or where the mucous membrane affected can be seen and reached, a solution of nitrate of silver (5—10 grs. to ℥j) or sulphate of copper (5 grs. to ℥j) should be used, with a camel-hair brush or as spray. Where ulceration can be seen, the Ung. Hyd. Nitrat. Mit. (Phar. Guy.) painted on, suffices. In cases of frontal pain, the inflammation having travelled up the infundibulum, volatile substances must be used; and I am accustomed to employ iodoform (30 grs.) mixed with starch and zinc powders (of each $\frac{1}{2}$ oz.). The proportion of iodoform must be altered so as to suit each patient's sensibility. In other catarrhal cases, generally strumous or syphilitic, iodoform is certainly

beneficial; and I usually add (following Trousseau) 5 grains of red oxide of mercury to each ounce of the "snuff." In impetiginous ozæna, softening of the scabs, which so plentifully block the nose, must be done with glycerine, and then the weak mercurial ointment be put on the exposed sores. If the case is seen early these will be within reach of a brush; if late, then after removal of crusts, weak stimulating lotions (sprays), as nitrate of silver gr. iij to ʒ j, are good.

The most hopeless (for the time) of all cases are the syphilitic necrotic cases; until the bone comes away, or is removed by operation, it is hard work even to keep the odour under; as deodoriser, iodoform is here useful.

Once I saw counter irritation do much good for the time in a girl æt. 13. Since two years of age she had had enlarged cervical glands that broke in 1878. At that time she had catarrhal ozæna with foul discharge and crusts. All this rapidly subsided for the three years that the glands remained open, but when they healed up, in 1881, the nasal discharge at once began to appear, although the child's general health had very much improved. Had the good result of the counter irritation been permanent, it would have suggested a seton in some obstinate ozæna cases, but hardly so as it was but temporary.

In constitutional ozæna local treatment must be combined with the general treatment adopted in such states.

ADENOID GROWTHS OF THE NASO-PHARYNX.

Although pathological enlargement of the lymphatic tissue of the naso-pharynx has not yet received the place it deserves in English surgical text-books, yet since Meyer's first description of it,¹ good accounts are to be found in writers specially devoted to the diseases of the larynx and pharynx.²

A mere outline of the more prominent symptoms is all that will therefore be given here, and the cases quoted will only be those of adults in whom the disease is far more

¹ 'Medico-Chirurgical Transactions,' vol. liii, p. 191, "Adenoid Growths in the Naso-Pharynx," W. Meyer, of Copenhagen.

² See especially Cohen, 'Diseases of the Throat,' New York, 2nd edition, 1879.

unusually met with than in the young. Cohen,¹ indeed, remarks (p. 258): "Most of the cases seen by myself have been in young adult males. In one instance I have had occasion to operate on two adolescent sisters. I have never seen it in advanced life." Meyer puts the most frequent age as between 5 and 25—rather a wide range! but believes it to be more frequent before ten years of age than afterwards. It is, as far as I have seen, most likely to be *complained of* as a pathological condition of young adults, for obvious reasons; yet if *systematically examined for* at all ages, irrespective of clinical symptoms, Meyer's statement would very likely be found quite correct. Whenever I have seen it in children it has been when examining for something else, and not because any definite symptom of its presence was visible. It will thus be seen that increased interest attaches to at least two of the cases here given, aged 50 and 40; they are the oldest I have met with either in practice or recorded.

The *pathology* of the disease is merely a hypertrophy of the normal lymphatic (adenoid) tissue found in the mucous membrane of the pharynx. When high up in the naso-pharynx it goes by the name of "adenoid growth of the naso-pharynx," but when placed lower down and within view from the mouth (though here it is probably ætiologically different), it becomes "granular sore throat," "chronic lymphadenitis of the pharynx," "Parson's sore throat," with about twelve other aliases.

A very good description of the minute and histological anatomy of the naso-pharyngeal lymphoid tissue and its hypertrophy is to be found in Cohen's work, already referred to, where he quotes largely from Luschka. The result of all microscopical observations is to show that the hypertrophied nodules are lymphoid tissue. In two of my own cases which I have examined (one being that of the woman æt. 40), the growths from the naso-pharynx, where prepared as microscopic sections, are perfect examples of this tissue.

Woakes, in his 'Deafness and Noises in the Head,' ed. 1880, speaks of the growths as papillomatous. There is no sound reason whatever given for such an assertion; it is quite unsupported and only mentioned here as a caution.

¹ Loc. cit.

The hypertrophied tissue is as soft as velvet, easily torn, and very vascular; accompanying it is a chronic catarrh of the mucous membrane, and hence the muco-purulent foul nasal discharge.

When adenoid growth of the naso-pharynx gives rise to symptoms, they are usually of sufficiently definite character to enable the affection to be diagnosed without physical examination, if the surgeon has once seen a case, otherwise the patient is likely to be passed over as merely the possessor of a common cold. It may not unfrequently be detected, in the drawing room, in young people when singing—there being an absence of clear ring in the voice that no amount of practice or vocalisation can get rid of. Mild, perhaps, in such cases, this symptom, when aggravated, causes a marked absence of nasal resonance—a “dead” pronunciation, as it has been called—owing to the blocking of the posterior nostril: and hence the patient comes for relief from chronic nasal stuffiness. Without actual stoppage of the nose the voice loses its clearness from the soft growth acting as a damper in the nasal “resonator.”

There is a sense of fulness in the throat, with a feeling of something there which must be swallowed or hawked up, and which is often then streaked with blood; and a constant discharge down the pharynx of muco-pus, which is worse in the morning, and at times gives rise to a morning sickness. When in bed, first one and then the other nostril gets blocked, and then always the one toward the pillow. There is a dry, unsatisfactory feeling in the nose, to relieve which the patient is constantly wringing the organ, even producing bleeding and excoriation. The patient when not talking keeps his lips apart, and of a morning complains of great dryness from having had to breathe all night through the open mouth.

A digital examination of the naso-pharynx through the mouth is more satisfactory than the rhinoscopic, and reveals either a velvety granular growth from the posterior wall (rarely elsewhere) of the naso-pharynx, very soft and easily wounded, or a larger growth, that has been compared to a number of rather small leeches hanging on to the mucous membrane. The former condition is the more frequently found. The examining finger will bring away always

some blood-stained mucus, and often the translucent millet-like grains, of which the growth is composed.

On examining with a good light the wall of the pharynx below the level of the palate, extension of the disease downwards may (but not always) be seen; but a thick, tenacious discharge trickling down is so common as of itself to suggest adenoid growth above, even if not otherwise indicated.

Deafness, due either to the growth having affected the opening of the Eustachian tube or to accompanying catarrh, is so commonly seen that Meyer summed up the diagnosis thus tersely by saying "a deaf patient who breathes through his mouth has probably adenoids."

Such a combination of symptoms as that glanced at above can point to but that one disease; for while polypus gives many similar indications, the influence of wet weather, the variability in the stuffiness, and the marked anterior discharge of water are unmistakable. *Ozæna* necessitates a foul smell; a "cold" has much anterior nasal catarrh, and anything but a dry condition of the nostrils; while phthisis, it is now seen, is not the only disease producing expectoration streaked with blood.

The vomiting, especially of a morning, requires more notice; and as illustrating this form of throat sickness, two cases are added which, though not strictly belonging to the class under consideration, are so closely connected with it that their mention will add to the completeness of the description.

Where the hypertrophy of adenoid tissue is confined to the naso-pharynx, the sickness, mostly of a morning, seems due solely to the fact of thick mucus trickling down the pharynx: the constant habit of hawking and spitting having increased, to a morbid extent, the naturally reflex sensibility to tickling the fauces.

When the disease is visible in the pharynx (whether also it exists or not higher up), the vomiting and retching seem to be the result of the extreme sensibility of the patches of adenoid growths themselves. When sometimes ulcerated, the pain of them is such as to produce dysphagia or even an utter impossibility to swallow anything hot or solid; an attempt to do this producing at once contraction of the constrictors, and hægismus and the return of the food by the mouth and

nose. The whole mucous membrane of the pharynx in these cases is very injected and abnormally sensitive; and not a few of them, as has been often observed, are in people of peculiarly "susceptible" natures.

In Cases 4 and 5 the ulceration occupied the salpingo-pharyngeal folds, and hence explained both the pain in the ear and the extreme agony caused by any attempt at swallowing. Zaufal¹ considers these folds are of more importance in closing the naso-pharyngeal cavity in deglutition than even the soft palate, and the pain accompanying their ulcerated condition would alone warrant the statement, apart from other observations.

That the enlargement of these folds of mucous membrane was due to lymphatic swelling in these cases was founded upon naked-eye appearances, together with the fact of there being adenoid growths elsewhere in the pharynx.

So extreme was the dysphagia in Case 5 that she had been sent for advice for probably malignant growth of the œsophagus.

Dysphagia, then, with the stabbing ear-pain are the prominent subjective symptoms of this form of sore throat.

The causation of these adenoid growths is very obscure, but being of lymphatic nature a scrofulous condition may be anticipated; yet, while undoubtedly scrofulous manifestations in physiognomy or in other ways may be seen at the same time in certain cases, especially in children, this is so seldom the case that the suggestion probably goes for nothing. Enlarged tonsils are also only an accidentally concurrent condition. The only constant peculiarity running through nearly all my cases (all but one) has been an unusually narrow naso-pharynx measured fore and aft; hence but comparatively little extra growth completely blocks the posterior nares.

Treatment.—This must be local, whatever general tonic treatment may be adopted besides. Astringents alone to the naso-pharynx, with carbolic acid to allay hyper sensibility, give relief but do not cure. "Scraping" the naso-pharynx, or actually cauterising the growths, is the only effectual means. The former is better if thoroughly carried out; the patients can mostly have it done without an anæsthetic, but they must be prepared to give several—perhaps half a dozen—sittings. The

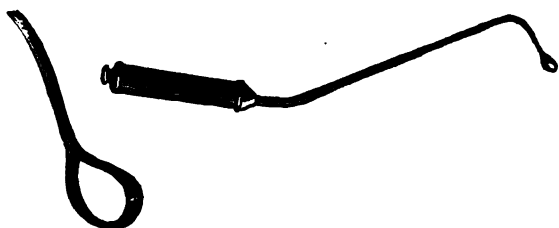
¹ Zaufal on "The Plica Salpingo-pharyngea," 'Archiv für Ohrenheilkunde,' 1879. Abstracted in the 'London Medical Record,' 1880.

actual cautery (galvanic) has been used, as also solid nitrate of silver; this last is useful as an aid after the scraping has been employed, and is very painful for some hours. The action of scraping does not seem to be only that of removal, for one does not see come away as much of the growth as afterwards disappears. The bruising action of the scraper starts inflammation, which seems quite as effectual in curing as the mechanical removal, even if not more so. The operation requires moderate care, as excessive inflammation, if excited, may bring the Eustachian tubes into trouble.

There are various shapes of forceps also used to evulse the growths through the mouth if large, or to crush them when small; but, except for the former purpose, scraping is more certain and easier to perform. I make use of Catti's forceps when I use any at all.

All operative interference with these growths induces sharp oozing of blood; but it never gives trouble, and syringing a little iced or salt water through the nose will arrest it, but even this is seldom necessary.

The operation of scraping may be done by a fine instrument introduced through the nose (Meyer's plan), or else through the mouth, which is more usual; but in this case the scraper must be bent to pass under the soft palate. I have had a scraper made, here figured, which answers admirably; it



being, in fact, a sharp fenestrated spoon set at the proper angle. It acts from above downwards, but some surgeons prefer working from side to side; for this a different instrument is required. A long forefinger-nail does the work very well; I never feel for adenoid growths without, if finding any, giving the naso-pharynx a good scrape before withdrawing the finger. It is, however, much more uncomfortable for the

patient, because of its bulk, than the metal scraper. If anæsthetised, the patient should be lying down with the head thrown well back, but if not he should sit facing the surgeon, with the head supported in the erect rather than the inclined position. With a rectangular tongue-depressor in the left hand, the right hand of the operator will be free to use the scraper, which should, as in all throat operations, be boldly and steadily introduced, without touching the tongue, beneath the soft palate. The scraping may be rapidly performed, the operation need not last more than ten seconds; not more than half this time, however, being often allowed by an intolerant and sensitive patient.

CASE 1. *Adenoids of naso-pharynx relieved by scraping mucous membrane.*—Ed. M—, æt. 50, out-patient, 26th July, 1880. A man of apparently average health; no evident constitutional defect.

Complains of stoppage of the nose, especially of the right side, during the last five or six years. Has had during that time a constant feeling of discomfort in the nose, with great dryness of the passages, and he tries to rid himself of this feeling by the constant use of his handkerchief and sniffing, as though to remove something there. Air passes through both nostrils, but with difficulty. The constant wringing of the nose has given it a red and chapped appearance, and so much is it a habit that whilst talking he, at least every minute, gets out his handkerchief and blows his nose, though nothing comes of so doing. He considers that he has a cold and complains much of the discomfort of the above symptoms. He has very marked dead pronunciation. As a general rule he breathes through the nose, and therefore does not carry the lips constantly apart. There is no anterior rhinorrhœa, but several times daily, and especially the first thing in the morning, he has to rid himself, by hawking and spitting, of much thick muco-purulent matter that trickles down his throat, which at times makes him feel sick.

Examination of anterior nares showed only a preternaturally dry and somewhat swollen and congested mucous membrane; no polypus, no ulcer. Digital examination of the naso-pharynx revealed a soft, velvet-like granular covering to all the

posterior naso-pharyngeal wall, into which the finger-nail easily passed, causing free bleeding, and bringing away small, clear, seed-like bodies resembling grains of sago.

The treatment commenced with a nasal douche (to be allowed to run out though the mouth) of glycerine and carbolic acid. This gave much relief to the feeling of dryness and made the patient more comfortable. On September 17th, 1880, he entered the hospital (Lazarus Ward) for a few days, and under chloroform I thoroughly scraped all the naso-pharynx; there was no difficulty experienced, the patient lying down flat with the head thrown somewhat backwards over a pillow. The bleeding was rather free, but syringing with ice-cold water at once arrested it. On his discharge, tannic acid was added to the lotion first ordered. October 18th, 1880, at the out-patients he was scraped again, without any anæsthetic. He now began to feel relief from his symptoms and to use his handkerchief less frequently. From this time to the end of the year he was again twice scraped, and also on January 10th, 1881, when I have this note: "Has been using Vapor Iodi, is much better, comparatively little growth (naso-pharyngeal) now; little hawking and spitting now; less discharge in the throat of a morning. Finds the Vapor Iodi gives much relief." The last note is February 7th, 1881: "Above (*i.e.* high up in naso-pharynx) there is nothing practically, but there is some still on level with soft palate; scraped again. Patient considers himself well. Scarcely any posterior (morning) discharge. Can breathe through nose." Has not been seen since.

CASE 2. *Strumous ozæna; adenoids of naso-pharynx; relieved by scraping.*—Harry S—, æt. 17, out-patient, 26th July, 1880. A lad of strumous appearance; has nose and upper lip swell in damp weather, when he easily catches cold. For a year has had stoppage of his nose at times, and there is much discharge down back of throat, which he hawks up. Never breathes through his nose, and keeps his mouth always slightly open; has complete dead pronunciation.

There is anterior and offensive rhinorrhœa with discharge of crusts. Of a morning his mouth is very dry from having breathed through it all night.

Examination anteriorly showed nasal mucous membrane,

especially of septum, red, vascular, and spongy, and easily bleeding; the swollen mucous membrane of the two walls nearly meeting at the narrow fissure between anterior and posterior nasal chambers. Digital examination showed small leech-like growths, pendulous from the posterior wall of the naso-pharynx, soft and easily bleeding.

The treatment here was in the main that of Case 1, with the addition of *Mist. Ferri et Quassia* ʒj, *ter die*. The patient had a most sensitive throat and resisted mechanical treatment, so that the scraping was never so thoroughly carried out as it should have been. Between July, 1880, and January 10th, 1881, the naso-pharynx was scraped four times, once under chloroform; and the last note of the case is "much better, breathes with comfort, and of a night also through nose."

CASE 3. Nasal polypus; removal of adenoids of naso-pharynx by scraping (From the report of Mr. H. E. RICHARDSON).—Mary Ann H—, æt. 40. First admitted into Mary Ward under Dr. Pavy, 13th May, 1881, and on the 27th was transferred to Martha Ward under Mr. Golding-Bird.

The patient was taken in for treatment on account of distressing eructations after taking food, uneasiness over pit of stomach, and interscapular aching pain. She gave a history of phthisis on both parents' sides, and she herself had been for four years subject to chronic bronchitis. A year ago had first had stuffiness in the nose, and five months later several nasal polypi were removed at St. Thomas's Hospital; some were (she knew) left behind.

The voice is natural save for a thick intonation due to the polypi.

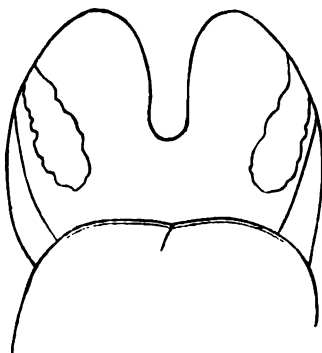
On May 20th, Mr. Golding Bird snared and removed some nasal polypi, and on the 27th she was transferred to Martha Ward for the operation to be completed, it being known that there was a large naso-pharyngeal polypus blocking the left posterior nostril. An attempt had been made on the 20th to remove it with the others, but without success. The following note was made in the surgical ward:—"At the first sitting, after clearing nostrils (of gelatinous polypi), a large naso-pharyngeal polypus was found blocking the left posterior nostril. Three attempts to "wire" it, Bell's fashion, failed. This

morning a dark gangrenous mass had been forced down to the anterior nostril, which proved to be the above polypus, destroyed by the first day's manipulation; it was easily removed now from the nostril. A further examination of the naso-pharynx showed velvet-like growth of adenoid tissue from its posterior wall; this was crushed with Catti's forceps, some being removed for microscopic examination."

Patient discharged the same day and has not been heard of since.

CASE 4. *Granular pharyngitis (localised) ; severe symptoms ; dysphagia ; relief by caustics.*—Ann B—, æt. 50, out-patient November 29th, 1880. A generally healthy woman; no constitutional defects. Two months ago seized rather suddenly with severe pain in the throat, very much worse on swallowing anything at all hard; the pain is referred externally to the angle of the right lower jaw. The pain has increased and now is most acute, flying up into the ears (right especially) as though a knife were being driven into it from within. Even when swallowing her saliva there is much pain, but the cutting sensation just mentioned is paroxysmal and induced by swallowing anything in the way of food. It is so intense now that she is unable to take enough to eat; she is losing flesh and looks very wan and ill. The pain markedly increased a fortnight since.

On examination, there is to be seen on each side of the



anterior wall of pharynx and partly under cover of the posterior

pillars of the fauces, a lineal growth of granulation-like tissue, the right one being ulcerated on the surface, raised a quarter of an inch from the level of the mucous membrane and running obliquely downwards towards the middle line from a point corresponding to the level of the pharyngeal end of the Eustachian tube. The upper end of each growth is too high to be seen; the lower extends nearly three quarters of an inch below the soft palate, and follows the line of the salpingo-pharyngeal fold. On touching the right one with a probe, a paroxysm of most violent pain was produced, flying up into the right ear; the left one was far less sensitive.

The treatment consisted of rubbing each spot with nitrate of silver (which was done with the greatest difficulty) and ordering an alum gargle and iron and quassia mixture. This was continued till January 13th, 1881, when the last note entered was "No pain; the swellings still visible but very slight." The patient ceased to attend.

CASE 5. *Granular pharyngitis; severe dysphagia; adenoids of naso-pharynx; treatment by crushing and scraping* (From the report of Mr. A. W. CLARK).—Mary Ann W—, æt. 27, out-patient June 13th, 1881. In-patient, Martha Ward, under Mr. Golding Bird, June 18th—22nd, 1881. A thin, anæmic, and cachectic woman, who last year had typhoid fever, since which time she had never been really well.

On June 13th she came to Mr. Golding Bird's out-patients, complaining of having for the last three weeks stuffiness in the right nostril and inability to swallow solids, because of great pain in the throat, and warm liquids as they at once came up through her nose. Talking was very painful and for two weeks she had subsisted on cold milk and arrowroot. There had been no anterior nasal discharge, no posterior that she knew of, no ozæna, no polypus.

On examination, the posterior wall of the pharynx was covered with a nasal discharge of muco-pus; this being washed away, there was seen a generally granular condition of the mucous membrane, and on either side, projecting forwards about a quarter of an inch, and running down in the direction of the Eustachian tube nearly parallel with the posterior pillars, was a club-shaped, red granular mass, as of hypertrophied adenoid tissue. It was not ulcerated but very tender; that on the

right side causing stabbing pain in the right ear when touched ; she had the same ear-pain also on swallowing.

The right nostril was found absolutely blocked ; the left free to air. There was very marked dead intonation. Digital examination showed large amount of granular adenoid growth on the right half of the posterior wall of the naso-pharynx ; only a little on the left.

She was ordered a gargle of alum and carbolic acid, and a mixture of iron and quassia.

On June 18th, when admitted for a few days, she was so much improved locally and generally that the same treatment was continued ; she could eat solids and only occasionally did liquids return by the nose.

21st.—The naso-pharynx was treated with Catti's forceps. Some of the growth coming away in the instrument. There was free bleeding but not much pain.

22nd.—“Can breathe through both nostrils. Her food is not returned through the nose. She can talk much better.”

The last note of the case is July 4th, 1881. “Came to out-patients' very nearly well ; all symptoms gone ; patches on throat scarcely visible ; can breathe well through both nostrils. Fine adenoid granulation being still felt, the naso-pharynx was again scraped.”

STATISTICAL ACCOUNT
OF THE
SURGICAL TREATMENT OF ANEURISM.

COLLECTED FROM THE HOSPITAL RECORDS.

By CHARTERS J. SYMONDS, M.S.

FULL reports of all the cases under treatment in the surgical wards have been made since 1866. From this source the following statistics have been collected, with the consent and approval of the surgical staff, and comprise the years 1866—80 inclusive. As the paper deals purely with the surgical treatment of the affection, only those cases submitted to such procedure are included. Thus are omitted, several aneurisms involving the large arteries at the root of the neck, with or without the aorta, also a case of iliac, and another of femoral aneurism, in which no treatment was employed; in one case owing to the patient suddenly disappearing, and in the other to the general arterial, and cardiac disease.

There are three cases of traumatic aneurism following a puncture of the vessel, two occurring in the radial and one in the temporal, which have been omitted. They were all slight cases and were treated by incision and ligature. Though the cases here collected include all those recorded in the volumes, it is

possible that a few may have been unreported, and that some reports may have been lost. The surgeons have been kind enough to examine the tables, and to add, where possible, points unreported, also to correct the results where these have not been clearly and fully recorded. In this way the accuracy has been increased, as well as the value of the record.

Cases which may have been treated in the medical wards have not been collected.

Excluding those mentioned above, eighty-two remain, and have been treated by the following methods:

Compression alone	42
„ followed by introduction of horse- hair into the sac	1
„ followed by Hunterian ligature	19
Primary Hunterian ligature	13
Distal ligature	3
By laying open the sac	3
Primary amputation	1
	<hr/>
	82

Examining first all the cases treated by ligature, the following table shows the particulars of each case. It is arranged in three divisions: first, all the Hunterian, next the distal, and, lastly, those treated by the old operation.

The particulars of the pressure employed in those cases, where this method preceded the ligation of the vessel, will be given when the compression treatment is considered.

TABLE I.
A. Cases in which the Hunterian method of ligature was employed.

Situation.	Sex, age.	Duration, size.	Previous preparation or treatment.	Artery ligatured and treatment of wound.	Kind of ligature.	Date of separation, in days.	Result.	Surgeon and reference.	Remarks.
1 Iliac, R.	M., 29	6 mos., hen's egg	Rest with ice 16 days	External iliac, wound open ¹	Silk	57	Cured	Bryant, 1868, 2	—
2 Ilio-femoral, L.	M., 60	3 mos., 2½ in. long	Rest and iodide 9 days	Do., closed, ² antiseptic	Carb. silk	—	"	Howse, 1879, 9	The wound closed over the ligature, the ends of which were cut short.
3 Common femoral, L.	M., 34	12 days, 1 in. in diameter	Rest and iodide 19 days	Do., open	"	18	"	Cooper Forster, 1879, 7	—
4 " femoral, R.	M., 28	1 month, 2½ in. long	Pressure 2 days	Do., closed	Catgut	—	"	Bryant, 1874, 5	—
5 Femoral, L.	M., 40	2 mos., 4½ in. in diameter	Rest 6 days	Do., closed	"	—	"	" 1877, 4	—
6 " femoral, R.	M., 45	12 mos., 5 × 3½ in.	Pressure 8½ hrs.	Do., closed	"	—	"	" 1874, 4	—
7 " femoral, R.	M., 33	18 mos., circumference 7¼ in. over other leg	" 10 hrs.	Do., open	Silk	—	Death	Cooper Forster, 1871, 21	From peritonitis 4th day.
8 " femoral, R.	M., 33	5 wks., 6 × 4½ in.	" 14 days	Femoral, open	"	16	"	Bryant, 1871, 24	From pyemia on 20th day. Sac opened on 13th day. Clot only evacuated.
9 " femoral, R.	M., 38	3 wks., 5 × 3 in.	Rest and ice 19 days	External iliac, open	"	48	Cured	Poland, 1866, 423	—
10 " femoral, R.	M., 39	2-3 mos., 3½ in. increase in circumference	Pressure 4 days	Do., open	"	23	Death	Cock, 1869, 22	Secondary hæmorrhage on 23rd, 28th, and 31st days; on last died.

¹ This expression means that one end of the ligature was led out of the wound.

² This signifies that both ends were cut short, and primary union attempted.

Case	Situation.	Sex, age.	Duration, size.	Previous preparation or treatment.	Artery ligatured and treatment of wound.	Kind of ligature.	Date of separation in days.	Result.	Surgeon and reference.	Remarks.
11	Femoral, R.	M., 27	2 mos., 3 in. increase in circumference	Rest and ice 3 days	Femoral, open	Silk	16	Cured	Cock, 1866, 424	—
12	"	L. M., 30	6 weeks	—	Do., closed	"	15	Death	Bryant, 1866, 428	From pyemia on 23rd day.
13	Femoral, L.	M., 37	16 days, 9 x 5 in.	Rest and ice 2 days	Do., open	"	?	"	" 1872, 221	From pyemia on 16th day.
14	Popliteal, R.	F., 54	6 mos., 2 in. increase in circumference	Rest 12 days	Do., open, anti-septic	"	23	Cured	Durham, 1879, 7	Rupture, with limited extravasation, few days before ligature. Hemorrhage on 67th day, requiring ligature.
15	"	R. M., 30	14 days, 2½ in. long	Pressure	Do., open, anti-septic	Carb. silk	30	"	Jacobson (Howse's vol., 1879, 11)	Separation hastened by weight.
16	"	L. M., 48	8 mos., 9 in. long	—	Do., closed, anti-septic	Catgut	—	Death	Howse, 1877, 9	Wound healed. Sac laid open on 32nd day. Died from chloroform.
17	"	L. M., 31	3 wks., reaches lower part of calf	Pressure 6 hrs.	Do., open	Silk	?	Cured	Durham, 1874, 7	—
18	"	R. M., 45	5 wks., "apple"	Emarch 3 times	Do., closed	Catgut	—	"	Bryant, 1877, 113	Gangrene of foot; amputation through the leg.
19	"	L. M., 38	6 mos., fills space	Pressure 3 days	Do., closed, anti-septic	"	—	"	Cooper Forster, 1877, 9	—
20	"	L. M., 27	2 mos., 4 x 3 in.	" 24 days	Do., closed, anti-septic	"	—	"	Howse, 1876, 5	—
21	"	L. M., 54	2 weeks	" 2 days	Do., closed	"	—	Death	Birkett, 1874, 12	From erysipelas and pyemia on 23rd day.
22	"	L. M., 60	4 mos., 4½ x 4 in.	" 2 days	Do., open	Silk	19	"	" 1874, 13	From gangrene on 19th day.
23	"	L. M., 26	12 mos., 3½ in. long	" 10½ hrs.	Do., open	Silk	?	Cured	" 1879, 14	—

24	"	R. M., 32	6 mos., 3 in. increase in circumference	"	10 hrs.	Do., closed, antiseptic	Catgut	—	Cured	Howse, 1872, 1	—
25	"	L. M., 32	5 wks., confined to space	"	36 days	Do., closed	"	—	"	Bryant, 1872, 24	—
26	"	L. M., 38	11 days, 4 x 2 in.	"	4 days	Do., open	Silk	15	"	Poland, 1871, 23	—
27	"	R. M., 27	4 mos., goose egg	Rest 3 months before and 1 after admission	Pressure 4 mos.	Do., open	"	?	"	" 1866, 426	—
28	"	L. M., 28	5 days, pigeon's egg	Pressure 4 mos.	Pressure and Esmarch	Do., open	"	27	"	Birkett, 1866, 427	—
29	"	L. M., 45	4 mos., hen's egg	Pressure and Esmarch	Pressure and rest 9 days	Do., closed	Catgut	—	"	Bryant, 1880, 65	—
30	Brachial, L.	F., 69	6 days, 4 in. in diameter	Pressure and rest 9 days	Brachial, open	Brachial, open	Silk	12	"	Poland, 1870, 17	—
31	Common carotid	M., 29	3 yrs., 3½ in. long	—	Common carotid, closed	Common carotid, closed	Whipcord	—	Death	Bryant, 1871, 23 ¹	From sloughing of sac and hemorrhage on 34th day.
32	Internal carotid, L.	F., 69	2 years	—	Do., open	Do., open	Silk	Firm on 11th day	"	Durham, 1872, 3	From increase of previous cerebral condition on 11th day; sac arose from first inch of artery, and was filled with coagulum.

B. Cases in which the distal method was employed.

33	Innominate	M., 33	18 mos., 2 x 2 in.	—	—	Subclavian, closed	Catgut	—	Relieved	Bryant, 1871, 22 ⁴	Slight hemorrhage on 14th, 15th, and 21st days. Sac was smaller and harder.
34	"	M., 40	9 mos., 4½ x 3½ in.	—	—	Left common carotid, closed	"	—	Death	" 1880, 63	Death from pressure of sac on 19th day.
35	Innominate and aorta	M., 58	12 months	—	—	Right common carotid, closed	"	—	"	" 1877, 1	Broncho-pneumonia on 12th day.

¹ A full report of this case will be found in the 'Medical Times and Gazette' for 1872.
² Quoted in Bryant's 'Practice of Surgery,' 3rd edition, vol. i, p. 447.
³ Ibid., p. 453.
⁴ Ibid., p. 455.

c. Cases in which the sac was laid open, and the vessel tied above and below.

Situation.	Sex, age.	Duration, also.	Previous preparation or treatment.	Artery ligatured and treatment of wound.	Kind of ligature.	Date of separation in days.	Result.	Surgeon and reference.	Remarks.
36 Femoral, R. M., 32		9 days	—	Femoral, open, antiseptic	Silk	Lower on 9th day	Death	Howse (Birkett's vol., 1873, 12)	Sac had ruptured six days before admission. The swelling reached the knee. There was intense pain. Secondary hæmorrhage 9th, 10th, 12th days; on latter he died. Had ruptured 14 hours before ligature.
37 " R M., 40 8 mos., 10 in. long			—	Do., open	"	10th lower, 15th upper	Cured	Birkett, 1867, 659 ¹	
38 Axillary, L. M., 43		4 yrs., 7 x 8 in.	Rest 3 months	Axillary, open	"	—	Death	Durham, 1879, 6	The sac had ruptured externally; death in a few hours.

¹ Published in detail in the 'Med.-Chir. Trans.' for 1867.

Proceeding to examine this table in the order of the subdivisions, the following plan exhibits the total result of the cases of Hunterian ligature.

TABLE II.

Vessel ligatured.	Cured.	Died.	Total.
External iliac	7	2	9
Femoral	14	6	20
Carotid	—	2	2
Brachial	1	—	1
	22	10	32

This shows a high mortality, especially after ligature of the femoral. On looking into the cause of death in each case, as shown in the next table, there will be found sufficient reason for reducing this death rate by two, thus bringing the proportion of deaths to one in four.

TABLE III.—*Showing the cause of death after the Hunterian operation.*

Case.	Aneurism.	Vessel ligatured.	Cause of death.	Day of death.
7	Femoral	External iliac	Peritonitis	4th day.
10	"	"	Hæmorrhage	31st "
8	"	Femoral	Pyæmia	20th "
12	"	"	"	23rd "
13	Femoro-popliteal	"	"	16th "
16	Popliteal	"	Chloroform	32nd "
21	"	"	Erysipelas and pyæmia	23rd "
22	"	"	Gangrene	19th "
32	Internal carotid	Common carotid	Cerebral softening	11th "
31	Common carotid	"	Sloughing of sac and hæmorrhage	34th "

In two of these cases the fatal result was not attributable to the operation. In one, the wound had healed, and the patient was in good health, but the aneurism remaining fluid, the sac was laid open, and just as the operation was completed the man succumbed to the anæsthetic. In the other, death was due to an increase of the cerebral condition existing prior to the operation. The patient had hemiplegia and aphasia, and after the

operation there was a temporary improvement, though the aneurism was situated just above the bifurcation of the common carotid. The softening was not limited to the distribution of the carotid.

It is noticeable that out of the twenty-nine cases of ligature of the main vessels of the lower limb, there is but one fatal by gangrene. In only one other did gangrene occur, and this was localised to the foot, the patient recovering after amputation through the leg. This case (No. 18) had been previously submitted to compression by Esmarch's bandage, and to the extreme congestion, and minute extravasations of blood which attended its application, the occurrence of gangrene was attributed, at least in part. There is also not a single case of supuration of the sac in these twenty-nine operations. This result followed in one of the carotid cases, and simulated acute tonsillitis. On the eighteenth day there was severe fever, and on the twenty-seventh the sac was laid open. This complication was preceded by secondary hæmorrhage, which ensued on the fifteenth day, and recurred several times; bleeding took place also from the sac, six days after it was laid open.

The proportion of deaths from pyæmia is certainly large, but it will be noticed, on referring to the table, that in none of these cases was the "antiseptic" method employed, and in one only the catgut ligature.

Secondary hæmorrhage proved fatal in one case out of the twenty-nine, and occurred in another, but was arrested by enlarging the wound and tying the upper end of the vessel. Catgut and antiseptic precautions were used, and a rapid recovery followed.

In all the cases, arrest of pulsation and diminution in size took place in the aneurism, and there was no recurrent pulsation.

The sac still remained fluid in one on the thirty-second day, and was treated as mentioned above. Amputation was proposed in the fatal case of gangrene, but in no other was this procedure necessary at the seat of ligature.

It is interesting next to inquire, how far the main causes of death are preventable, and to this question the first table gives a striking answer, showing how the mortality has diminished with the improved methods of treating wounds, and with the

use of the catgut ligature. Taking the Hunterian and distal operation only, or, in other words, those in which the vessel was ligatured in its continuity, thirty cases are available for examination. Those are included in which, either death was directly due to the existence of a wound, or in which the patient recovered.

Of the eight cases excluded, four died from other causes than those arising in the wound (Nos. 22, 32, 34, 35). One died from peritonitis on the fourth day, a result independent of the form of ligature, though perhaps not of the mode of treatment of the wound. The other three were treated by the old plan.

To this list may be added one other, in which the external iliac was tied with silk, the tumour proving to be a pulsating sarcoma. Death occurred from secondary hæmorrhage. In one of the "silk" cases (No. 2) the wound was dressed under the spray, the end of the ligature at the same time being cut short, and the wound closed.

Of these thirty-one cases, silk was employed in nineteen, catgut in twelve. There were six deaths after the use of silk, viz. three from pyæmia, and three from secondary hæmorrhage, and in one other this latter complication arose, but was controlled. So that, in these seven cases, the complication arose owing to the existence of the wound, which was prevented closing, on account of the form of ligature employed. This gives a mortality of 31·5 per cent. and 36·8 per cent. of complications.

In the twelve cases treated by catgut, one proved fatal from pyæmia, and in one other there was slight secondary hæmorrhage. This gives a mortality of 8·3 per cent. and 16·6 per cent. of complications.

Antiseptic precautions were used in four cases, and in the other eight the wound was closed, and not disturbed for several days. In some of the cases no second dressing was required. Neither of the cases in which a complication occurred was under the antiseptic method. Now, from these facts the conclusion seems to follow, that upon the closure of the wound the success of the case in great measure depends.¹ If this can be secured

¹ Mr. Holmes remarks on this point, "We have in the Hunterian operation a method, which almost always succeeds if the patient recovers from the operation," '*Lancet*,' 1874, p. 861, vol. ii.

without suppuration, the present statistics prove that the mortality will be greatly reduced. For, taking the cases fatal from all causes as enumerated in Table III, together with the one last introduced of pulsating tumour, it will be seen that, seven out of the eleven deaths were due to the wound remaining open. Excluding from this list No. 16, in which the wound had healed, seven deaths out of ten must be attributed to the open state of the wound, or a proportion of 70 per cent.

These numbers, though too few to decide the value of the catgut ligature finally, are sufficient to show that the use of this material, especially when combined with antiseptic precautions, is one means at least of reducing the mortality. If No. 2 be added to the cases where the wound was closed, though the ligature was of carbolised silk, the advantage of this method comes out more strongly.

It has been found by some that catgut is uncertain, that it does not hold long enough to ensure the formation of a good coagulum. The twelve cases here recorded exhibit its effects, three times on the external iliac, eight times on the superficial femoral, and once on the subclavian. In the ten successful Hunterian cases, there was never any return of pulsation.

In one case it will be observed that carbolised silk was used, and treated in the same manner as catgut. The wound healed readily, and in others it has closed by primary union. This completely meets the objection as to the uncertainty of catgut, and with a closed wound there is no fear of secondary hæmorrhage. To ensure this behaviour with the silk, the antiseptic method will probably be more essential than with catgut.

Some information also may be obtained from the table, as to the influence of previous compression upon the result of ligature. Taking still the twenty-nine cases of the Hunterian operation in the lower limb for examination, it will be found that there was no previous compression in eleven, and of these three died. The vessel ligatured in all the fatal cases was the femoral, the aneurism being femoral in one and popliteal in two.

Ligature followed compression in eighteen cases, with five deaths. In two the external iliac was tied, in three the femoral, and of the latter, in one the aneurism was femoral, in two popliteal.

Case No. 16, in which death occurred from chloroform, must

be considered as a failure only, reducing the mortality after primary ligature to two. From the successful cases after secondary ligature one, No. 18, must be counted as a failure, gangrene of the foot resulting.

This gives of failures—

After primary ligature . . . 3 or 27·27 per cent.

„ secondary „ . . . 6 or 33·3 „

Taking, for comparison with other published statistics, the cases of popliteal aneurism only, there are thirteen of secondary ligature with two deaths and one failure, and four primary with one death and one failure.

The percentage in the first is 22·07 per cent. of failures, and 15·46 of deaths, an average greatly in favour of previous compression. This result is, therefore, the opposite of that deduced from the total of twenty-nine cases. Mr. Hutchinson,¹ who speaks in favour of the effect of previous compression, gives four deaths out of ten cases of primary ligature; and, according to Mr. Holmes, five deaths out of nineteen cases of ligature after compression, or a failure in the latter of 26·31 per cent. Mr. Holmes² gives, as the result of his tables, a failure of 29·54 per cent. in secondary ligature, against 19·48 per cent. in the primary.

The sum of these three tables gives a total of eighty-three ligatures following compression with twenty failures, and 112 of primary ligature with twenty-one failures, a result still showing, as Mr. Holmes says, “to any one who trusts implicitly in figures” previous compression to be unfavorable.

Mere death-rate, which forms the larger proportion of failures, does not seem sufficient ground upon which to test the influence of previous compression. For in these twenty-nine cases only two deaths out of the seven are attributable to the ligature,³ the rest being due to complications that might attack any wound. Again, the pressure was in all the fatal cases except one, of short duration. The case in which gangrene occurred was compressed for nine and a half and ten hours on two days; too short a time, apparently, for any serious injury to be inflicted upon the artery or the vein. The longest

¹ ‘Medical Times and Gazette,’ Nov. 29, 1856.

² ‘Lancet,’ May 1, 1875.

³ Nos. 10 and 22.

period was fourteen days, the others ten hours, two days, and four days. There was not time in any of these cases, for exhaustion to have occurred, sufficient to determine a fatal result. Though, therefore, the death-rate is against previous compression, yet, in examining the mortality more closely, it seems that this treatment has had little to do with the failures in the present list of cases. The frequency of suppuration in the sac, and return of pulsation, besides gangrene, would appear to be the proper grounds upon which to consider the influence of previous compression. It does not seem easy to say, whether the causes leading to gangrene existed prior to compression or not. A more careful dissection of the fatal cases, with special regard to the condition of the arteries below the ligature is necessary, before it can be decided in what cases gangrene is likely to follow, even upon primary ligature. If it were known, for instance, that the vessels had lost much of their elasticity, would not *a priori* reasoning suggest at least a short trial of compression? In two of the cases, pressure was maintained for thirty-four and thirty-six days; both recovered after ligature. In another case placed in the compression list, treatment was kept up for six months; subsequent ligature became necessary, and the man recovered. These points are mentioned to show, that other facts than mere death-rate are necessary, in forming an opinion upon the influence of previous compression.

The other three cases of Hunterian ligature call for no further remark as regards treatment.

The old operation of laying open the sac and securing the vessel above and below, was performed in three cases. In two the aneurism was femoral, in one axillary. The last case died shortly after the completion of the operation. The aneurism had ruptured through the skin, and a good deal of blood was lost prior to the operation. In the other two cases the rupture was recent, in one of six days', in the other of about twelve hours' duration. In both a catheter was introduced into the vessel as an aid in dissecting it out. Both suffered from secondary hæmorrhage, which proved fatal in one, the other¹ recovering after ligature of the vessel immediately above the original seat of operation.

¹ A detailed account is given in the 'Med.-Chir. Trans.' for 1867.

Of the three cases of distal ligature the one that recovered received distinct benefit. Two years later he was readmitted with increase of symptoms, and refused to have the carotid tied.

Cases treated by compression.—The following table shows the gross result of this method, calculated from which, the success is 53·2 per cent. Further on it will be shown that the success was really greater.

TABLE IV.

Aneurism.	Total.	Failed.	Cured.
Iliac	2	1	1
Femoral	7	4	3
Popliteal	46	21	25
Subclavian	1	—	1
Axillary	1	—	1
Brachial	2	1	1
Ulnar	1	—	1
Carotid	1	1	—
Innominate	1	1	—
	62	29	33

The next table gives particulars of all the cases submitted to compression only, with one in which horsehair was afterwards introduced into the sac. There are here forty-three cases. The remaining nineteen, in which ligature was subsequently employed, have already been tabulated, and particulars of the compression treatment will be presently given.

TABLE V.—Cases in which compression alone was employed.

Situation.	Sex, age.	Duration, size.	Form of pressure.	Mode of application.	Duration of treatment.	Result.	Reference.	Remarks.
1 Iliac and popliteal, same limb, R.	M., 40	Iliac 2 years, 2 x 1½ in. Popliteal 9 mos., 3½ x 2½ in.	Instrumental (tourniquet)	Complete control of common iliac under chloroform for 5½ hours; after 11 days of iliac and femoral 9½ hours	19 days	Both cured	Hilton, 1870, 9	No improvement from first pressure. This case is considered as an iliac aneurism only. The popliteal is not included in the statistics.
2 Iliac, L.	M., 41	24 days, 4 x 3 in.	Instrumental	Intermittent control for 8, 6 and 19 10 hours, with 6 and 10 days' interval; under chloroform	19 "	Death	Birkett, 1869, 24	Death from pneumonia 6 days after last compression
3 Femoral, R.	M., 34	5 weeks, 5 x 3 in.	Digital	Complete	9 hrs.	Cured	Howse, 1880, 42A	Pulsation was arrested in 5 hours; the remaining treatment to ensure success.
4 " L.	M., 31	3 weeks	"	Complete control	8½ "	"	Forster, 1876, 8	—
5 " ?	M., 37	8 weeks, 4 x 4 in.	"	Complete control	7½ "	"	Howse, 1879, 10	—
6 Popliteal, R.	M., 33	2 months, "large"	"	Complete for 8 periods averaging 10 hours, with intervals of rest	21 days	"	Durham, 1879, 6	—
7 " R.	M., 41	5 weeks	Digital, instrumental, Esmarch	Complete and partial 59 days; Esmarch 4 hours on 60th day, followed by instrumental for 15 days	75 "	"	Forster, 1878, 6	Improvement from the first; rapid after Esmarch.

8	R. M., 38	1 month, 4 in. long	Digital	Complete for 2 periods of 12 36 hrs. hours, with 12 hours interval	"	Howse, 1877, 8	—
9	L. M., 38	Pigeon's egg	"	Complete for 2 periods of 12 3 days hours, with 1 day's interval	Slight improvement	Do.	This aneurism noticed on admission; ligature refused.
10	L. M., 31	2 months, egg	Flexion alone, and with instrumental	Complete for 2 hours; partial 3 days	Cured	Durham, 1876, 2	—
11	L. M., 30	1 month, fills space	Digital and instrumental	Complete for 9 and 13 hours on 2 days; partial for 11 hours on 6 days	"	Bryant, 1876, 7	—
12	L. M., 35	3 mos., 2 x 1½ in.	Digital	Complete	"	Forster, 1876, 9	—
13	R. M., 48	5 mos., 2½ x 1½ in.	Flexion, digital, instrumental	Partial by first for 7 days; complete by second for 14 hours; partial by third 9 days	"	Forster, 1876, 10	Improvement from the first; unable to bear digital.
14	L. M., 40	3 mos., small	Instrumental	Complete on 2 days for 6 29 hours; partial, with intervals, for 27 days	"	Forster, 1874, 8	The pulsation feeble from the first.
15	R. M., 47	4 mos., 3 in. long	"	Intermittent arrest 3—7 hours on 4 days; complete and partial for 6 days after 3 days rest; then, after 6 days rest, complete in 12 hours	"	Durham, 1873, 17	There was considerable pulsation before last compression.
16	R. M., 38	3 mos.	"	Complete for 9 hours; rest for 4 days	Improved	Forster, 1873, 9	Readmitted 2 months later with return pulsation.
17	L. M., 38	—	Digital and instrumental	Complete for 8, 6, and 7 hours on 3 days, with 8 and 10 days interval	Slight improvement	Do.	—
18	R. M., 47	5 mos., pigeon's egg	Instrumental	Complete for 9, 11, and 15 hours, with 4 and 6 days interval	Cured	Birkett, 1873, 13	Popliteal on other side cured by ligature 7 years before.

No.	Situation.	Sex, age.	Duration, size.	Form of pressure.	Mode of application.	Duration of treatment.	Result.	Reference.	Remarks.
19	Popliteal, R.	M., 31	2 mos., hen's egg	Digital	Complete for 8, 16, and 16 hours on 3 days	3 days	Cured	Bryant, 1872, 23	See 'Med. Times and Gazette' for 1872
20	"	L. M., 35	10 days, 2 in. long	Flexion and instrumental	Complete under chloroform on four occasions for 4, 6, 4, and 6 hours; the rest partial between these times	55 "	"	Forster, 1872, 11	—
21	"	R. M., 27	1 month, ?	Flexion, instrumental, digital	Partial and complete in three periods, with 7 and 5 weeks interval; digital complete	Active for 3 mos., rested for digital 25 and 4 12 hrs.	Pulsation twice arrested, finally permanent	Birkett, 1872, 7	He had aortic disease, and left against advice.
22	"	L. M., 40	1 month, 2½ × 1½ in.	Instrumental, digital by patient	Complete for 12 hours; rest partial and intermittent	Discharged in 27 days	Cured	Durham, 1870, 6	—
23	"	R. M., 33	6 weeks, 3 × 2 in.	Instrumental	Complete	7 hrs.	"	Hilton, 1870, 10	—
24	"	R. M., 41	10 mos., swan's egg	"	Partial	9 days	"	Hilton, 1869, 21	—
25	"	R. M., 36	17 mos., pigeon's egg	"	Complete	7 hrs.	"	Birkett, 1869, 10	—
26	"	R. M., 44	3 mos., 2 in. diam.	"	Complete for 10 hours, when pulsation ceased; reappeared in few hours; then 9 hours on 2 days; after 4 days rest intermittent complete for 12 hours on 2 days, with 1 day's interval	10 days	"	Forster, 1868, 1	—

27	"	L. M., 29	8 weeks	"	Partial	20 "	"	Hilton, 1868, 4	—
28	"	L. F., 26	5 mos., barton's egg	Flexion, instrumental, digital	The first for 3 days; the next 37 days partial; digital complete for 27 hours on 41st day	41 "	"	Hilton, 1867, 660	—
29	"	L. M., 34	3 mos.	Instrumental	Partial	11 "	"	Birkett, 1867, Same as No. 25, 661	
30	"	L. M., 32	5 mos., "fat"	Digital and instrumental	Digital complete for 24 hours on 1st and 20th days; 16 days of rest and 2 instrumental	20 "	"	Bryant, 1867, 662	See also 'G.H.R.,' ser. iii, vol. xiv, p. 241. The man himself subsequently cured an aneurism in the right popliteal space by 44 hours pressure. Ligature recommended.
31	"	L. M., 24	1 day, very small	Instrumental	Partial	7 "	Slight improvement	Cock, 1866, 425	
32	"	R. M., 65	18 mos.	Digital	Complete on 2 days for 16 and 11 hours, with 1 day's interval	3 "	Cured	Lucas, 1880, 29	Arrested for a time after first compression.
33	"	R. M., 42	5 wks., 3½ in. long	—	Esmarch ¼ hour; immediate pressure, complete for 7 hours	7 hrs.	"	Golding Bird, 1880, 29	—
34	"	L. M., 44	2 mos., "orange"	Instrumental	Partial	6 mos.	No improvement	Forster, 1876, 3	He had subclavian and right popliteal aneurisma. Femoral ligatured 1½ years later by Dr. Poole.
35	Popliteal at bifurcation, L.	M., 33	3 weeks, 2½ in. increase in circumference	Digital, instrumental	Complete by first for 11 and 12 hours on 2 days, and by instrumental for 8, 7, 9, 10, 8, and 7 hours, with 7 days interval between 2nd and 3rd day; then partial by patient for 4 hours on 6 days	22 days	"	Bryant, 1873, 11	18 feet of horsehair introduced. See Bryant's 'Practice of Surgery,' 3rd edition, vol. i, p. 456.

Situation.	Sex, age.	Duration, size.	Form of pressure.	Mode of application.	Duration of treatment.	Result.	Reference.	Remarks.
36 Popliteal at bifurcation, R.	M.	? Hen's egg	Digital	Complete for 12 hours; after a fortnight again complete for 9 hours	16 days	Cured	Howse, 1880, 43B	Perchloride of mercury given for the fortnight.
37 Posterior tibial, upper part, R.	M., 40	3 years, 3 in. long	Flexion	Complete arrest on 2 days for 1 hour; on third day partial, combined with digital, by patient	3 "	"	Bryant, 1869, 16	The leg was kept flexed for 12 days longer. See 'Med. Times & Gaz.,' 1872.
38 Carotid, L.	F., 55	9 weeks, "small"	Digital	Complete under chloroform, on distal side	5 hrs.	No improvement	Jacobson, 1879, 1A	—
39 Innominate	M., 55	3 mos., 2 x 1 in.	"	Complete under chloroform to carotid	9 "	Death	Forster, 1872, 9	—
40 Subclavian, R., 3rd part	M., 44	2 years, 1 x $\frac{1}{2}$ in.	Digital, with pad	Complete, with a few hours occasional interval	96 "	Cured	Poland, 1868, 1	—
41 Axillary, R.	M., 36	3 mos., 3 in. in diameter	"	Complete under chloroform for 4 days, 4 $\frac{1}{2}$ and 8 $\frac{1}{2}$ hours on 2 days, with 2 days interval	4 days	"	Forster, 1872, 10	—
42 Brachial, R.	M., 31	1 month, 2 $\frac{1}{2}$ x 2 $\frac{1}{2}$ in.	Instrumental and digital	The first complete 3 hours, 17 hrs. then alternately with digital 14 hours	17 hrs.	"	Birkett, 1870, 16	—
43 Ulnar, L.	M., 50	5 weeks, 3 in. in diameter	Digital, flexion with pad	Complete for 5 hours and 10.15 hours on one day	15 "	"	Poland, 1872, 1	—

¹ By "complete control" is understood that an attempt was made to arrest altogether the flow through the main artery. There is frequently during the change of compressors a varying flow permitted. "Partial" means that the flow, though at times completely arrested, at others is more or less free.

The proportion of success is slightly better than that stated above, as in four cases the pressure was of short duration, other conditions demanding the ligation of the vessel. Particulars of the compression in these cases are given below (Nos. 6, 23, 24, 17), under the heading of "Cases afterwards ligatured," where it will be seen that, the pain and increase in the size of the tumour in two, the previous diffused condition of the aneurism in another, and, in the fourth, the irritable state of the patient, were reasons for discontinuing the trial of compression. In one it is probable a cure would have resulted, in the others the aneurism was probably already too advanced for treatment by pressure. Omitting these four cases the success is 56·89 per cent.

The duration averages a little over thirteen days. The shortest time is seven hours, though in No. 3, all pulsation was arrested in five. The method of summing up the duration of treatment, includes the periods of rest between the various trials. From this cause, in many instances it appears long. To give, however, as accurate an account as possible of the time during which active treatment was employed, a special column has been devoted to this. In examining the records, carefully reported as they have been on the whole, here and there it has not been possible to make out the exact duration of treatment. This applies chiefly to those cases where the weight or tourniquet has been left in charge of the patient. In such cases there may be a little inaccuracy, a day or two being unaccounted for in the report. The time in these is, however, of less moment than in those counted by hours.

Before passing to the unsuccessful cases, it may be well to single out the popliteal aneurisms. There are forty-six. Of these twenty-five were cured, a percentage of 54·3. Three of the cases demanding early ligature were popliteal; deducting these, the success becomes 58·1 per cent.

Mr. Hutchinson gives forty-six cases with twenty-four cures, a percentage of 52·1. Mr. Holmes gives sixty-six cures in 124 cases, a percentage of 53·2. The similarity in these results is remarkable.

Of the twenty-nine unsuccessful cases—

19 were followed by ligature with fourteen cures. In

1 twenty feet of horsehair were introduced into the sac.

7 remained improved or unimproved. And
2 died.

Details of the five unsuccessful cases of ligature have already been given. The effect of the horsehair was to produce "almost complete consolidation of the tumour."¹ The patient had valvular disease of the heart, and died of arterial pyæmia. Two cases died: one (No. 39) was an innominate aneurism under the care of Mr. Cooper Forster, by whose kindness I am enabled to give the details. The first sign of disease was pain under the clavicle, in the right side of the neck, and back of head, three years before admission. For three months he had noticed a tumour at the root of his neck on the right side. When first noticed it was as large as a walnut, and when he first came under observation, there was a pulsatile tumour behind the inner third of the clavicle and the sternum. The right pupil was smaller than the left, and he had ptosis. He was admitted on the 3rd of April, 1872, and for seven days the diet was restricted. Five days later chloroform was administered, and the carotid compressed by means of a pad held in the hand from 11 a.m. to 8 p.m. For the first two hours a good deal of chloroform was used, after that a little was sufficient. There was much coughing, with intervals of sleep. At 8.10 p.m. the breathing nearly ceased and became stertorous. At 9 o'clock artificial respiration was resorted to, with galvanism, considerable improvement resulting. No movement was observed to take place in the left arm and leg after 9 p.m. The next day there were "spasms" on irritation, and a strong pulsation in the aneurism. He remained in a comatose condition till 4 p.m. on the 21st, when he died: just five days and five hours from the commencement of pressure. Fifteen months before he was admitted, he had a suddenly occurring left hemiplegia, from which he slowly recovered.

At the inspection, made by Dr. Goodhart, the aneurism proved to be innominate. It involved the whole artery and the root of the carotid. The aortic orifice was enlarged to the size of a five-shilling piece. The aorta itself was bulged in places. The aneurism had absorbed the origin of the sterno-hyoid and sterno-thyroid muscles, but the bone was uninjured. The sac was as big as an egg, and was nearly full of laminated old

¹ Bryant's 'Practice of Surgery,' 3rd ed., vol. i, p. 456.

coagulum, like soaked brown paper. The subclavian seemed to be quite intact, at least half an inch of it existed between the sac and its first branches. Both hemispheres of the brain were blood stained, as if from decomposition, but equally so. Vessels not particularly full on either side. On slicing the brain, patches of hæmorrhage were found scattered through the cineritious substance; some of them were scarcely larger than a pin's head, others were aggregated together in masses, equal in size to a hazel nut, the intervening brain substance being softened. The smaller hæmorrhages were all in the cineritious substance, and the larger, of course, could not be determined by the naked eye to be limited to this part, but they followed the outlines of it and probably were so. In the white matter the bloody points were numerous, but there was no hæmorrhage. The extravasation was more considerable on the left side than on the right; there was none on the lower surface towards the base. The arteries at the base were rather thick walled, but not markedly diseased, and contained no emboli. The bronchial tubes were intensely reddened, and contained a yellowish puriform fluid. Those at the base of the right lung markedly dilated, with intervening portions of the lung airless.

The other fatal case occurred in the practice of Mr. Birkett, who is good enough to permit its publication here. This was an iliac aneurism. The first symptom was a sensation as of the "bursting of a vein" in the groin twenty-four days before admission. He had no pain, swelling, or inconvenience for eighteen days, at the end of which time, a pulsating and painful swelling appeared in the groin. When admitted, February 5th, 1869, the skin was tense and congested and the thigh cedematous. On the 7th the common iliac was compressed for eight hours (under chloroform), the pulsation being at times completely arrested. At the end of this time the pulsation was not diminished. Two days after, there was pain and numbness in the leg. On the 18th the pulsation being as forcible as on admission, the pressure was reapplied (for six hours), and repeated eleven days later for ten hours. The next day it is noted that there is slight pulsation in the most prominent part of the tumour. He became drowsy, the temperature rose on the 17th to 102°, and pneumonia was diagnosed. He died March 3rd, six days from the last application of pressure.

The sac was found to extend from two inches above, to five inches below Poupart's ligament, and to be nearly filled with soft coagulum. The abdominal wall was ecchymosed and infiltrated with inflammatory material. The common iliac was surrounded by adhesive lymph, firmly attaching the lymphatic glands to it. No injury was sustained by the intestine, nor was there any other lesion which could be attributed to the pressure.

These are the only cases in which any untoward result followed the application of pressure, and the details of the post-mortem examinations are especially interesting. Although pulsation continued in the iliac case, the appearances suggest that a cure would have resulted.

In seven cases this form of treatment failed, and was not followed by any other. Short notes of the treatment in each case have already been given in the table; a few additional particulars of interest are here recorded. The numbers refer to Table 5.

No. 9.—The aneurism in this case was small. The man himself had not observed it. When compression was applied the sac had existed six weeks; no improvement resulted though the opposite side was easily cured. It is not improbable that, as this aneurism was detected early, the communication with the artery was large in proportion to the sac. This patient refused to submit to ligature.

No. 31.—The pulsation was noticed by the man after a fall, in which he sustained a scalp wound. There appears to have been no previous pain, though the disease must have existed some little time, at least a few days. He refused to have the artery tied.

No. 21.—The symptoms were first noticed after wrestling. He had also aortic valvular disease. There was a gradual improvement in the aneurism, and after six weeks, pulsation ceased altogether, and he was discharged "cured." Before he left the hospital, however, it was noticed that slight pulsation occurred when he put the foot to the ground, and also when a splint was applied to straighten the knee. Twenty-five days after the cessation of pulsation it recurred, and was again arrested for four days. Subsequently the aneurism increased in size, but again compression failed. The man now left against

advice, having been under treatment more or less actively for six months.

No. 16.—In this case pulsation ceased four days after the compression was discontinued. Three days later some pulsation, thought to be due to enlarging collaterals, was noticed. A month later, when discharged, there was no pulsation, nor was there any in the tibial arteries.

There is no mention in this, or in the preceding case, of the condition of the swelling. Two months after, he was readmitted with return of the pulsation, making the time three months from its cessation. It is not stated how soon the recurrence took place, or what use he made of his leg. Compression was again resorted to, with the result of diminishing slightly the size. After a stay of three months he was discharged, with both aneurisms pulsating, no improvement ever having occurred in the left (No. 17). Further treatment—probably ligature—was proposed, but was not permitted. The return pulsation in this case took place at least a month, and probably more, after its cessation. No mention is made of cardiac disease.

No. 34.—The duration in this case is two months. Four weeks previously he sprained his knee, but at first suffered no inconvenience. During his stay of six months, the pulsation was often controlled, and considerable improvement resulted, so that, notwithstanding the subclavian aneurism and the other arterial disease, he was for some months able to work.

For the remainder of this case I am indebted to Dr. Wordsworth Poole, of Sidcup, who, in reply to an inquiry, has kindly furnished the particulars, and has generously allowed me to make use of them.

In 1876, about a year after his discharge from the hospital, the aneurism threatened to burst, when Dr. Poole ligatured the superficial femoral, using catgut, and closing the wound. Pulsation ceased entirely, and never returned. The progress of recovery was very slow, the man being, as Dr. Poole says, "upstairs" for four months, during which time there was severe pain down the leg. "After this, but not till then, the tumour diminished, and gradually got down to a size rather more than half what it was before operation." He was able to work as a cowkeeper a year after the operation, and continued this employment, with intervals of unfitness for work, till

within three months of his death, which occurred August 28th, 1880. This was attributed to aortic aneurism, but no inspection was permitted. Dr. Poole, who saw him a week before death, says "that the right popliteal and subclavian aneurisms, did not increase or trouble him at any time." Another interesting feature in this case is that, eighteen months before death, or twenty-eight months after ligature, "the tumour grew again, till it was as large as ever, but no pulsation of it as a whole could ever be felt, though the cone still contained fluid, and the beating of a small artery could be detected over one part of the swelling at a distance from the cone." This case is another success after the use of catgut, and has *not been included* in the previous statistics. The spray was not used.

No. 88.—No ill result followed the treatment.

It has still to be explained why pressure failed to cure these, and the other nineteen cases. But it will be noticed that in four out of the seven, the patients refused further treatment, and therefore the cases count as total failures, without the opportunity being afforded of ligating the artery, in those where it was thought a suitable operation.

*Details of the compression in the cases afterwards ligatured.
The numbers refer to table I.*

No. 4.—Common femoral. Complete by weight for ten and fifteen hours on two days. The aneurism was fusiform. No improvement followed, but it rapidly disappeared after the ligature of the vessel.

No. 7.—Femoral (common?). Complete by tourniquet to external iliac under chloroform for ten hours. No improvement. The sac was close to Poupart's ligament.

No. 10.—Femoral (two inches below Poupart's). Partial by tourniquet for four days. Pulsation continued forcible.

No. 6.—Femoral. Partial arrest for eight and a half hours, on the next day for one hour. The aneurism had been rapidly enlarging for three weeks, and had suddenly increased six days before admission. The projection of the tumour was diminished by the pressure, but the pain was very severe. The pulsation was not reduced.

No. 23.—Popliteal. Digital, complete for ten hours. The

tumour was harder, and the pulsation less. He was irritable, and could not bear the repetition. The other vessels and the heart were healthy.

No. 24.—Popliteal. Digital, complete, with interruptions for ten hours. The aneurism was very large and extended up the inner side of the thigh. The pressure reduced the pulsation and pain for a day, but the next and following days the pain was severe, and the swelling increased in size, the skin becoming tense and red. The artery was ligatured on the third day after compression, and was followed by a rapid cure.

No. 17.¹—Popliteal. A tourniquet was used for six hours. The aneurism had ruptured before admission, the calf was brawny and œdematous. Compression of the afferent vessel arrested pulsation, but did not diminish the size. The man had a to-and-fro aortic murmur. The low position of the swelling suggested an embolic origin. There was some increase of the tumour during compression.

No. 15.—Popliteal. Digital, complete for twelve, six, and six hours on three days, with a day's interval. Pulsation continued, but the tumour was harder. The patient had right hemiplegia of recent origin, from which he was gradually recovering. No cardiac disease could be discovered.

No. 19.—Popliteal. Digital, complete for six and a half, and eighteen and a half hours on two days, with a day's interval, and nine hours by tourniquet immediately after the second digital trial. Very little benefit resulted.

No. 20.—Popliteal. During eleven days there was pressure by tourniquet for twelve hours on eight days, digital for fifteen hours on two days, digital and instrumental for twelve hours on one day, with a day's interval. During another ten days, the patient controlled the tourniquet for ten to twelve hours daily. After fifteen days' rest digital pressure was employed for about twelve hours on two days. Three days later the artery was tied. This patient was a dispenser, and, having the "dangerous" amount of knowledge, wished to avoid the ligature. There was no other arterial disease, and the heart appeared healthy.

No. 18.—Popliteal. This case has already been fully

¹ This and the three preceding, are the cases referred to above, where other conditions than failure of pressure demanded ligation.

reported by Mr. Bryant, in his work on 'Surgery,' 3rd ed., vol. i, p. 447. Gangrene of the foot followed the use of Esmarch's bandage.

No. 21.—Popliteal. Compression by the weight, by the tourniquet, or by the finger, for ten and fifteen hours on two days, with complete arrest. "Perhaps harder" is the only result stated. Though the report does not positively state it, two days' further instrumental may be assumed, two days after which the ligature was employed.

No. 21.—Popliteal. Complete by tourniquet for an average of nine hours on four days. Once it was continued for twenty hours. After a day's rest a weight was employed during eleven days, with intervals of two and four days. Then digital by patient for ten days, followed by complete arrest for twelve hours by digital means. Lastly, a tourniquet was controlled by the patient for another nine days. It was harder, but the pulsation was forcible. The whole period was thirty-six days. Rapid cure followed the ligature.

No. 8.—Popliteal. Intermittent arrest by weight on five occasions, with intervals of one, two, and seven days. The pulsation diminished for a time, but increased seven days later. The groin was sore.

No. 26.—Popliteal. Complete for fourteen hours by digital method, and tourniquet intermittently so, for three days. Tumour enlarged and became soft, and suppuration was feared. The patient was exhausted. Recovery after ligature was rapid.

No. 29.—Popliteal. Complete digital on five occasions for an average of fourteen hours, with intervals of two, two, seven, and nine days, during the last of which the patient kept a weight on the artery. Pressure maintained once for twenty-four hours. After a day's rest Esmarch's bandage was applied for one hour, followed by weight compression for eight hours by the patient. No improvement resulted, so that after an interval of five days the artery was tied. Compression of the afferent artery arrested the pulsation, and caused great diminution of the tumour. He had aortic disease. There was pain afterwards along the course of the artery or vein, with œdema of leg and pyrexia. For a full account of the case, see 'Lancet' for July 9th, 1881.

No. 28.—Popliteal. Partial by tourniquet for four months,

followed by complete digital for fifty-five hours. Little or no change. A year previously this patient had an aneurism in the opposite space cured in four weeks, and for this reason he wished to delay the resort to the ligature.

No. 22.—Popliteal. Complete, by weight for nine hours and a half, and by digital for ten hours, on two separate days, with two days' interval. There was no improvement. In six days the swelling increased in size, the skin became tense, and there was great pain. Two days later the vessel was ligatured.

No. 30.—Brachial. Flexion one day, digital thirteen hours on the next, after which the pulsation was arrested for a few hours. On its recurrence, though the sac was thickened, ligature of the brachial was immediately undertaken.

The case requiring primary amputation was a woman, aged sixty-two, who ten weeks before strained her knee. It immediately swelled, and five weeks after pulsation was noticed in the popliteal space. When admitted blood was oozing through the skin in several places, and gangrene was threatening. The case was considered by Mr. Cock, who performed the operation, to be one of ruptured artery. No record of the examination of the vessel is preserved. The case is in Mr. Cock's volume for 1870, No. 15.

AN INQUIRY
INTO
THE PHYSIOGNOMY OF PHTHISIS
BY THE METHOD OF
"COMPOSITE PORTRAITURE."

By FRANCIS GALTON,¹ F.R.S.,
AND
F. A. MAHOMED, M.D.

THE doctrine of diatheses, or what is often called "temperaments," in other words, the belief that certain physical conformations indicate predispositions to certain diseases, has always held so prominent a place in medicine from the earliest ages that it is unnecessary to dwell upon its history or its present position at any length. Of late years this doctrine has been repudiated by many of our most able teachers, though on the other hand it still receives the powerful support of some of the most distinguished and experienced of our physicians. So that what heretofore has been generally accepted has now become a much disputed question. The objections that have been raised against the doctrine by those of what may be called the new school are chiefly these: that it is founded on the utterly false and erroneous doctrine of "humours" held by physicians in the dark ages; that it is therefore only a relic of false traditions; and lastly, that it is not supported by any modern scientific observations, and that the statements of

¹ Though it would be difficult wholly to disentangle our respective shares in the inquiry, I must at least give the entire credit of the following memoir to Dr. Mahomed.—F. G.

"general impressions" made in support of it are those of impressions prejudiced by traditional beliefs.

In reply to these objections it may be said that the facts which the earlier physicians observed were probably correct enough, and that it was only their explanations and theories that were wrong; thus they may have observed certain facts in connection with the physical characteristics of individuals in association with certain diseases, and then sought to explain them by their false theories; the facts may nevertheless remain true.

The objection that this doctrine is only supported by personal impressions still holds good, and it is with a view to put it to the test of exact, and as far as possible unprejudiced investigation, that the following observations have been made.

Probably no diathetic types are more commonly recognised, either rightly or wrongly, than the so-called tubercular and strumous; both of these, but more especially the former are held, by those who believe in them, to play a prominent part in phthisis, inasmuch, that persons presenting the physical characteristics attributed to these diatheses are said to be especially predisposed towards this disease. It has appeared to us that this belief might be put to the test by means of the method of 'composite portraiture;' in short, that we might be able to ascertain whether there are any facial characteristics common to any large proportion of cases of phthisis.

In the first place, it was necessary that we should accumulate a large number of photographs of patients suffering from this disease, and with this view we obtained permission from the physicians of Guy's Hospital to photograph any patients coming under their care; the authorities of the hospital were also good enough to place the photographic studio at our disposal.

We began our work in January, 1881; by March we found that the progress was too slow, and that we must extend our field of operations in order to get a larger supply of patients. We therefore sought and obtained the permission of the physicians and the governing bodies of the Brompton and Victoria Park Hospitals for Diseases of the Chest, to photograph a large number of their phthisical patients. We would take this opportunity of expressing our gratitude to the authorities of these hospitals for the great facilities they so freely afforded us

for carrying out our observations, and also our most sincere thanks to the physicians and resident medical officers (Dr. Hicks, of Brompton, and Dr. Humphry, of Victoria Park) for their very kind co-operation and assistance in our work. When all did so much, and so willingly, it would be invidious to mention those who were able to contribute most; but when we say that from the out-patient rooms and the wards of these hospitals we were supplied with about 400 cases, and that, in nearly all, the cards to be afterwards described were filled up by the physicians in the case of the outpatients, and by the resident medical officers for the inpatients, some idea may be formed of the labour entailed upon these gentlemen.

Our endeavour has been throughout to protect ourselves from any charge of a prejudiced selection of cases or distortion of facts; we therefore supplied those kind enough to help us with cards on which the chief details of the cases could be briefly recorded, by making a "tick" in the appropriate space; a copy of these cards is here inserted, the method having proved at once simple and convenient.

Hospital.		
PLEASE PHOTOGRAPH BEARER.		Initial of Physician.
Name.	Age.	Date.
		1881
EXTENT OF DISEASE	ONSET OF DISEASE	
Advanced	Insidious	
Moderate	OR PRECEDED BY	
Slight	Severe hæmoptysis	
DURATION OF DISEASE	Bronchitis	
Chronic (over 3 yrs.)	Pneumonia	
Medium (1—3 yrs.)	Pleurisy	
Brief (under 1 yr.)	Syphilis	
HEREDITARY TAINT	Gout	
Strong	Alcoholism	
Some		
None		
Remarks		

We asked the physicians to send us all cases of well ascertained phthisis occurring in either sex within the limits of

fifteen and forty years of age. These limits of age were fixed, partly because the faces between these ages are more fairly comparable, and partly to exclude the more evidently acquired phthisis of advanced age.

Mr. Galton then engaged the services of Mr. Mackie as photographer, who has had large experience in rapid photography, having been employed professionally for some years in photographing, for the use of the authorities, the prisoners at the Pentonville Convict Prison. He was often able to secure for us twenty and thirty portraits from the out-patients in the course of an hour or two, and we were enabled to obtain the number we required during the months of April and May.

We now found at our disposal 442 portraits of patients suffering from phthisis, of whom 261 were males and 181 females. They were obtained as follows :

From Brompton—	Males .	140	
	Females .		116
„ Victoria Park—	Males .	36	
	Females .		42
„ Guy's	Males .	85	
	Females .		23
		—	—
		261	181
		—	—
Total	.	442	

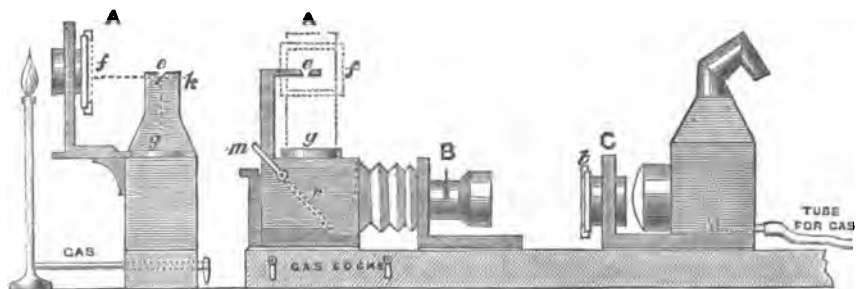
For comparison with these we next proceeded to photograph 100 male and 100 female patients, taken without selection from the wards and out-patient rooms at Guy's, none of whom were suffering from phthisis ; these we propose to use as a standard to represent the average of the population of the same class, but suffering from diseases other than phthisis.

During the process of making composites, we received some valuable assistance from Mr. George Turner, who came to our help when we were much pressed for time and made several composites for us, besides assisting in the selection of faces out of which they should be formed.

Before discussing the results obtained, it will be well to say a few words in explanation of the method followed. Composite portraits may be described as *pictorial averages*, they

are independent of the fancy of the operator, just as numerical averages are, though like them, they may be vitiated by errors of calculation or of manipulation. Composite portraits, the method of producing them, and their adaptation to various purposes, have already been described by one of us on several occasions;¹ but it may be desirable for the benefit of our present readers to give an account of the process. They are

APPARATUS FOR MAKING COMPOSITE PORTRAITS.²



- A The body of the camera, which is fixed.
- B Lens on a carriage, which can be moved to and fro.
- C Frame for the transparency, on a carriage that also supports the lantern; the whole can be moved to and fro.
- r The reflector inside the camera.
- m The arm outside the camera attached to the axis of the reflector; by moving it, the reflector can be moved up or down.
- g A ground-glass screen on the roof, which receives the image when the reflector is turned down, as in the diagram.
- e The eye-hole through which the image is viewed on g; a thin piece of glass immediately below e, reflects the illuminated fiducial lines in the transparency at f, and gives them the appearance of lying upon g,—the distances f k and g k being made equal, the angle f k g being made a right angle, and the plane of the thin piece of glass being made to bisect f k g.
- f Framework, adjustable, holding the transparency with the fiducial lines on it.
- t Framework, adjustable, holding the transparency of the portrait.

¹ "Composite Portraits," by Francis Galton, F.R.S., 'Journ. of Anthropological Inst.,' 1878 (reprinted in 'Nature' and in the 'Photographic News,' and translated in the 'Révue Scientifique.') "Composite Portraiture," by the same author, 'Photographic Journal,' June, 1881 (reprinted in the 'Photographic News,' July 8th and 15th). The process there described was the one adopted in the present inquiry. See also "Generic Images," by the same author, in the 'Proceedings of the Royal Institution,' to which some autotype reproductions of composites are appended.

² This woodcut is borrowed from the 'Photographic Journal,' June, 1881.

made from the individual negatives by throwing upon a photographic plate, disposed in a special form of camera, the images of several negatives in succession, an equal fraction of time being given to each, instead of the whole time to one. Thus, if a plate requires 200 seconds of exposure, and it is wished to make a composite of ten individuals, the image of each negative will be thrown upon the sensitised plate for twenty seconds. To obtain a clear result it is, of course, necessary that the chief features of each negative should fall as far as possible upon the same points of the plates submitted to exposure; this is attained by carefully adjusting the image of each in respect both to scale and position. The camera is furnished with appliances to enable the operator to do this, consisting of numerous adjustments, by means of which the various images are brought into exactly similar relations with certain fiducial lines thrown on the focussing glass. The latter consist of one vertical line for the median line of the full face, passing down the middle of the nose, and two horizontal ones, the upper to pass through the pupils of the eyes, the lower across the mouth. The image of each negative is enlarged or diminished as may be necessary, to secure that the distance from eyes to mouth may be the same in all cases. It is then rotated and shifted up or down and sideways, until the upper of the two horizontal lines intersects the pupils and the vertical one divides the face equally. The outlines of the face are entirely disregarded and left to take care of themselves. As it is necessary that the plate to be exposed, having been once put into position, should not be shifted until the close of the operation, and as each negative has to be focussed in succession, the composing camera is made with a horizontal focussing glass on its roof, as well as, or instead of, at the back. By means of a swinging reflector, let down at an angle of 45° with the top of the camera, the image produced by the lens is thrown upwards on to the horizontal focussing glass in the roof, where, by means of a camera lucida, the fiducial lines and certain tinted dots, illuminated by a standard illuminator (marked 'gas' in the diagram, but more properly a candle), are seen as if marked on the horizontal focussing plate, and to these lines the image is adjusted, as already described.

By the use of the standard tinted dots the gas illumination

of the negative is controlled, so that the image of each portrait has equal intensity, and therefore contributes equally to the result; the errors that would be produced by the varying densities of the negatives are thus in great measure obviated. When the image has been exactly adjusted, and the proper illumination has been obtained, the reflector is raised and the image allowed to fall upon the sensitised plate; this process being repeated with each of the component negatives.

A single plate that has been exposed to several negatives yields what is called a *composite*. Several of these composites may in their turn be exposed to another plate under similar conditions, as if they were ordinary negatives; the result is called a *co-composite*. Several of these co-composites may be combined to produce a co-co-composite, and so on.¹

With this brief account of the process, which is fully described in the papers already referred to, we may pass to the consideration of the photographs obtained. On looking over the individual portraits of the patients suffering from phthisis, one is first struck with the absence of those characteristic faces which we expected to find among them. With the exception of a few who were very severely ill, the faces did not seem to differ much from those of any group of ordinary patients, indeed, there seemed nothing characteristic about them. They were shown to many physicians, many of whom expressed their surprise at the absence of characteristic faces. We were inclined to accept this at first as a distinct answer in the negative to the question, Is there a tubercular diathesis? But after much sorting and arranging into groups, and after combining the individuals, so as to test the similarity of their features, certain results began to unfold themselves. Clinical facts were first taken as guides for grouping; thus the cases of "advanced disease" were grouped, but gave no result beyond well-marked emaciation (Pl. I, fig. 10, and Pl. II, fig. 22). The rapid cases of "brief duration and advanced disease" yielded no characteristic type, nor was anything very definite obtained at first from those in whom the "hereditary taint" was "strong." The one of us least likely to be prejudiced by preconceived notions, dealt with these latter cases single-handed and without consultation with the other.

¹ Composites and co-co-composites are positives and require to be reversed before printing from them.

Concerning them Mr. Galton writes as follows:—"Fifty-six cases (among the women) were recorded by the medical officers as having a strong hereditary taint of phthisis, and it is of these alone I now speak. On first examination of the collection of portraits, I was chiefly struck by their diversity, but after familiarising myself with them and sorting them tentatively in various ways, I began to perceive what seemed to be natural groups, leaving comparatively few that I could not classify. I made composites of each of these groups; there were eleven of them, containing on an average five components each, one only had as few as three, and one only as many as nine. I then sorted the composites and found that they fell into two main divisions, not, however, separated by any abrupt line of demarcation. In the one division there were six composites of, on the whole, thirty-six portraits, and in the other there were five composites of twenty portraits in all. The first division had blunted and thickened features, the second had thin and softened features. I then made a compound composite of each of the two divisions (Pl. I, figs. 4 and 6), and finally I threw both divisions into a doubly compound composite (co-co-composite, Pl. I, fig. 5) to form the general average. I need not stop here to speak of the precautions taken in doing this, further than to mention that the groups were always "weighted" in exact proportion to the number of their constituents, as by giving thirty-six seconds exposure to the co-composite of the first division against twenty seconds to that of the second division, when forming the general average.

"The trustworthiness of the final result must be estimated on the same principle as if we had been dealing with numerical averages. That is to say, we may rest content whenever the averages derived from two large subdivisions of any group resemble the general average as nearly as is needful in the case under consideration. I think this result has been fully reached in the present case, for notwithstanding that the divisions have been made so as to contrast as strongly as possible, their composites (Pl. I, figs. 4 and 6) resemble very nearly that of the general average (Pl. I, fig. 5). It is therefore obvious that if the eleven primary composites were divided into any other pair of groups, the co-composites of each of these two groups would have a yet more close resemblance to each other, and to the

general average also. I have indeed made some trials which amply confirm this view. Therefore, as far as concerns the female patients between the ages of eighteen and forty in London Hospitals who have phthisis, with a strong hereditary tendency to the disease, I have no doubt that any future inquirer who deals as I have done with not less than fifty cases, will arrive at an ideal face almost identical with that which I have produced." The truth of this last remark as regards all cases of phthisis, has been strikingly corroborated by our further investigations, as will be seen by comparing with this result the three other co-composites of phthisis, containing about fifty cases in each (Pl. I, figs. 7, 8, and 9). The two co-composites (figs. 7 and 8), taken absolutely without selection, are almost identically the same face, while fig. 9 is composed of two opposite types of faces—the narrow ovoids and broad faces with coarse features; and this has produced a rather stronger face than either of the others, a nearer approach to the non-phthisical patients.

These observations were made while one of us was away from London; on his return it at once became evident that what have been described above as the two types, the one with blunted and thickened features, the other with thin and softened features, closely coincided with the two types constantly described by physicians as the "strumous" and "tubercular." Proceeding now to carefully sort our patients under these two heads, and to put our selections to the test of combination in composites, we soon obtained very striking and highly characteristic faces.

Reviewing the whole of our results, two important conclusions may be adduced, and these may be given as a preface to the consideration of the plates in detail. It appears that the method of composite portraiture may be employed to obtain two different and equally advantageous results. First, by throwing into one a large number, say fifty different faces, taken without any selection whatever, we can obtain an average of them all; but this presents no features or expressions characteristic of what may be called secondary types; such a result is an excellent method of obtaining the broad average as to the general proportions of the face, the average shape of the lower jaw, the average delicacy or coarseness of the features, and the

average amount of emaciation, &c. Secondly, it is possible by taking very carefully selected faces to form a composite face having certain characteristic features; in making such a face the introduction of a few which are not strictly admissible into the group readily effaces the characters sought for in the composite, and as but few faces can be found which closely correspond, the larger the number employed the more does the result approximate to the general average face. This method of combining specially selected faces, is in itself an excellent test of the correctness of the selections made. If the result obtained has lost the special characters sought for, we may be sure that the faces selected were ill assorted; always, however, bearing in mind that the larger the number of faces introduced, the greater the probability of reverting to the general average. This method fails to obtain for us so typical a face as may often be seen in a single individual, yet it tests the accuracy of our opinions as to the general similarity of several selected faces.

Plate I is composed almost entirely of general average faces, both male and female, and the uniformity of the results is very noticeable. Figs. 1 and 3 each contain fifty patients, all suffering from diseases other than phthisis, taken without selection, and chiefly from among the out-patients attending Guy's Hospital. The results, over which the operator can have no voluntary control, are remarkably alike; yet they consist each of fifty entirely different people, no single person occurring in both. With these compare figs. 5, 7, 8, and 9 in the same plate; each of these contains about fifty cases of phthisis, and again the results are wonderfully alike, although another face is arrived at. A certain selection has been made in figs. 5 and 9; the former contains, as above mentioned, fifty-six cases, in all of whom a strongly-marked hereditary taint existed, and the resulting face has distinctly more delicate features, and is a narrower ovoid than figs. 7 and 8. Fig. 9, on the other hand, is composed of two opposite extremes; it was formed by combining a composite of selected narrow ovoids, and one of selected broad faces with coarse features, and contains most of the components of figs. 29, 30, 31, and 32, Pl. II. The result is a face standing midway between the very delicate fig. 5 and the broader faces and coarser features seen in figs. 1 and 3.

From a consideration of these, we are undoubtedly justified in saying that the average of phthisical faces gives more delicate features, an apparently lighter lower jaw, and an altogether narrower face than the average of other diseases. Probably in some measure this result is due to the greater average emaciation of these patients than that of those suffering from other diseases. But emaciation will not always alter the general outline of the face; this is well shown by Pl. I, fig. 10, a composite of eleven cases of phthisis in whom the disease was far advanced; in this face the results of emaciation are well shown in the deeply sunken eye, the hollow cheeks, and thinly-covered lower jaw, but the face nevertheless is not by any means a "narrow ovoid." A critical examination of fig. 1 will show, however, that emaciation in such a face would take away much of the "heavy-jowled" appearance of the lower part of the face, and would thin the nose and lips greatly, bringing it nearer to the phthisical type.

Pl. I, fig. 2, was obtained by selecting all the narrow ovoid faces among the hundred female patients not suffering from phthisis; it contains fifteen individuals. It may be compared with figs. 29 and 30, Pl. II, which contain nine and twelve individuals respectively, or twenty-one in all; these are the selected narrow ovoids occurring among our total number of 181 phthisical women. Pl. I, fig. 2, will be found to be very closely similar to Pl. II, fig. 29. We have, then, the unexpected result of 15 per cent. of the non-phthisical women giving this narrow ovoid face, and only 11.6 per cent. of patients with phthisis presenting it. It must be remembered, however, that many of these fifteen may hereafter develop phthisis, for several of them were young women suffering from those ill-defined functional disorders which often precede it. We may also find another explanation of this result in the fact that we are here dealing with phthisis among the lower classes, and that with them phthisis is probably much more often an *acquired* disease than what is called a *constitutional* one. Yet, allowing due weight to these considerations, the fact still remains well established that no larger proportion of peculiarly narrow ovoid or delicately-formed persons could be selected among those suffering from phthisis than among the ordinary female population; on the other hand, the general average of each class proves the

phthisical women to have the more delicately-featured and narrow faces.

We may now examine the men, and we shall find very similar results. Pl. I, fig. 11, contains 100 patients suffering from diseases other than phthisis; it is a co-co-composite of figs. 13, 14, 15, and 16, which are co-composites containing twenty-five in each, being composed each of five composites having five individuals in each composite. This subdivision was only employed to check an error more readily and make repetition of a single group easier and shorter if necessary. On the other hand, fig. 17 is a co-co-composite of 206 cases of phthisis, it contains figs. 18, 19, 20, and 21, each of which contains fifty (except fig. 18, which contains fifty-six); these four are co-composites, each containing five composites of ten individuals suffering from phthisis, and taken without selection. Fig. 18 was made entirely from patients under treatment at Guy's, and the average severity of these cases is usually greater than those treated at the hospitals specially devoted to chest disease. A comparison of these groups gives much the same result as in the case of the females. The phthisical composites are evidently much narrower and more delicate faces than those suffering from other diseases. The same exact similarity is not at once apparent in the series of male phthisical faces as in the female, chiefly on account of the variable growth of hair, but still a strong resemblance is to be traced between them. It may be remarked that fig. 13, containing non-phthisical persons, presents a more delicate face than figs. 14, 15, and 16, and closely approaches to the phthisical type; an explanation of this is to be found in the fact that it chanced to contain six out of the thirteen narrow ovoid faces contained in fig. 12, or, in other words, three times as many narrow ovoid faces as in either of the remaining three, supposing the rest are equally distributed.

Pl. I, fig. 12, contains thirteen narrow ovoid faces selected from the 100 cases 'other than phthisis,' and it may be compared with Pl. III, fig. 33, the co-composite of fifty-one narrow ovoid faces selected from the 262 males suffering from phthisis, and also with the two female narrow ovoid faces (Pl. I, fig. 3, and Pl. II, figs. 29 and 30). As in the case of the females, the two male narrow ovoids chiefly differ in the degree of emaciation visible, this being well marked in the phthisical cases and

absent in the other diseases. The proportion of narrow ovoids in each class among the males is the reverse of that among the females, for we find only 13 per cent. among the cases 'other than phthisis,' and 19.46 per cent. among the phthisical patients if we include all the fifty-one cases contained in the plate; but at least six of these, we shall hereafter find, ought to have been excluded, for they do not belong to the "narrow ovoid" class. This would bring the number down to forty-five, which would give almost exactly 17 per cent. If we add together the percentages of the narrow ovoids in both sexes we find that the cases 'other than phthisis' give 14 per cent., while the phthisical cases have 14.3 per cent., in short, they are to be found in equal numbers both among the phthisical and non-phthisical patients. Let us here emphasise the fact that we are now comparing phthisis with *other diseases*, and not with the healthy population, and these observations would seem to show that a delicate person may fail in many ways besides becoming phthisical, and that a delicate narrow ovoid face, may mean liability to other diseases not necessarily tubercular.

Turning to Pl. II, fig. 22 is a co-composite of forty-two cases of advanced phthisis, all of whom showed in their faces the ravages of the disease; it contains the six composites, figs. 23, 24, 25, 26, 27, and 28, each containing seven individuals. The co-composite closely resembles fig. 33, the co-composite of narrow ovoids, and both of these in their turn closely approximate to the phthisical type seen in fig. 17 and its components; yet the components of fig. 22 are strongly dissimilar.

Fig 23, one of the components of fig. 22, is a most typical, perhaps the most typical phthisical face, yet the individuals contained in it were in no way selected, except for the severity of their disease; they were taken merely in the order in which they chanced to be photographed. It is interesting to compare this face with figs. 29 and 30, and some of the composites in Pl. III; in several of these instances the same face is very nearly arrived at. In this face the large projecting ears are very noteworthy; they are noticeable in several other composites, and in many of the component faces; these, taken together with a narrow mouth, often open, a short and small chin, a small and narrow lower jaw, make together an often-recurring face in

phthisis. A very typical face of this nature is seen in Pl. III, fig. 34, No. 101. This face is one of the components of fig. 23, and lends to it much that is characteristic.

We have remarked that the method of composing faces is a good test of their real resemblance, that is, the more closely allied they are to each other the better composite will they produce; this is well exemplified in figs. 27 and 28, but especially in the former, in which the faces, having been taken without selection, have very imperfectly combined. While by adjustment the central features have been exceedingly well blended, yet the main outlines of the various faces remain very distinct, and at least five out of the seven it contains can be traced around the chin and ears, especially on the left side.

Figs. 29 and 30 are two composites of narrow ovoid faces, and have been already referred to. Their components are arranged adjacent to them; they are the nearest approaches we could find to the so-called "tubercular type," which seems singularly rare or much modified among the lower classes of the population. An attempt has been made to arrange them in two groups, containing a higher and a lower type of face, but the results are very similar.

The components of fig. 30 are chiefly characterised by the large ears, the narrow, open mouths, with prominent upper teeth, and short, small chins, which have been mentioned as forming a frequent type in phthisis. No. 608, the last portrait on this plate, was also included in this group. This face presents the narrow ovoid outline, but the coarse features and broken nose of fig. 32.

Figs. 31 and 32 are the direct converse of the narrow ovoid just described. In these we find the broad faces, heavy lower jaws, short upper lips, thick and rather up-turned noses, often with a depressed bridge, which are characteristic of what is called the "strumous diathesis." When we examine a group of the most degraded of this type, as seen in fig. 32, we cannot but recognise that we are dealing with such features as those which characterise syphilis. This view seems well borne out by the five faces at the bottom of this plate, and in the composite produced by them. If we compare fig. 31 and its components with them, we can readily trace a close similarity between these and the more degraded ones in fig. 32. Possibly one or two

generations have sufficed to effect the change, so that the deformed and ill-formed faces, the direct products of disease, when sufficiently diluted, may give rise to the comely and attractive face seen in the composite, fig. 31, and in one or two of its components. In this way we may often observe the disappearance of eccentricities and deformities, and that return to the average type, by which alone the maintenance of the race is possible.

In Plates III and IV, the male patients having the same characteristics as the females in Plate II, have been selected and combined. They have required greater subdivision on account of their larger number and the growth of hair upon the faces. Among the narrow ovoids the selection has not been sufficiently critical, and several have been admitted which might with advantage have been excluded; the final co-composite, Pl. III, fig. 33, would then have been more typical. As it is, the outlines are somewhat ill-defined, and the face scarcely as narrow as it should be. In this group, moreover, numbers have increased the difficulty of arriving at a type; it contains fifty-one components and its tendency is to revert to the general average, as may be seen by comparing it with fig. 17, which it much resembles.

We would remark that the nine composites forming the components of fig. 33 are only stages in the production of the co-composite. The faces in each composite have not been selected for their resemblance to each other, but merely as belonging to the "narrow-ovoid" class. They have been chiefly taken in the order in which they were photographed, except that in the first three figures the hairless faces have been put together while in the next two those wearing hair have been combined; all contained in these first five composites were patients at Brompton, those in the next three at Guy's, and those in the last at Victoria Park.

The component composites of fig. 33 are more characteristic than the co-composite itself, though not so typical in many cases as they should be. Fig. 34 somewhat resembles fig. 30, and one of its components, No. 101, bears some resemblance to No. 578 in the female group. These are the two most typical faces in either, and partake more largely of the characteristic features of the groups than any other single faces; they therefore more

closely resemble the composites. It may be argued that they have contributed too large a share to each, and possibly this may be true. They both exhibit in a striking degree the characteristics of the lower so-called "tubercular type" already described. Out of the components of fig. 34, No. 160 should have been excluded.

In fig. 35, No. 192 might have been omitted on account of the irregularity of his features, but his ears are highly characteristic of the semi-idiotic or degraded type.

Figs. 36 and 37 are striking faces and good results, but in fig. 38 none of the faces are very characteristic, though all belong to the narrow ovoid class.

Fig. 39 has been ruined by the admission of Nos. 7 and 11; No. 7 can only have been admitted by mistake, and No. 11 owes his admission to his emaciation and not to his original conformation; for the broad strong angles of his lower jaw are characters the reverse of what is noticeable in the other faces on this page. No. 6 also might have been excluded with advantage. Fig. 39 may be compared with fig. 18, and a strong resemblance traced between them and accounted for by the fact that among its fifty-six components fig. 18 includes all the components of fig. 39, as it does also those of fig. 40. From the latter figure Nos. 20 and 36 and perhaps No. 15 might have been omitted. Indeed, had the last two figures been merged into one and only allowed to include Nos. 9, 13, 28, 24, and perhaps 23, a far better result would have been obtained, both in it and in the final result (fig. 38). Time, however, did not allow us to make these alterations, when, on arranging these plates the errors of selection were discovered; in some measure it is perhaps desirable that the corrections should not have been made, for it will allow our readers to see both the strength and weakness of the process, that is, its mechanical accuracy and the check it makes on the selection of faces, and, on the other hand, the failures and misleading results obtainable by bad selection. This question of selection is still further emphasised by fig. 41 (Plate IV), which contains a very ill-assorted collection of faces; long and short faces, and the broad lower jaw of No. 212, being all mingled in terrible confusion, giving the many outlines to the face obtained in fig. 41, which almost every one of its components can be traced.

Fig. 42, on the other hand, gives a very good result, except as regards its mouth.

Fig. 43 on Pl. IV, and the composites (figs. 44 to 48) out of which it has been formed, contain twenty-seven individuals selected as possessing broad faces with coarse features. In this group a difficulty arises from the fact that there is a mixed class, the representatives of which among the men are chiefly included in these composites; they have a narrow ovoid face, but with coarse and thick features, as in the case of No. 608, fig. 32, among the women, who was also included in the narrow ovoids of a lower type. If this work was being done again it would be well to put these in a class by themselves. Many of these faces are included in fig. 44, and the result has been to obtain a decidedly narrow ovoid face; the same is true of fig. 47, and these two faces having been admitted to the co-composite fig. 43, have done much to destroy its typical characters, though it still remains a well-marked contrast to fig. 33, and it much resembles the corresponding female composite fig. 31.

Fig. 44 contains eight individuals, namely, those on a line with it in the plate. We have already remarked that they present much in common, but that their faces are mostly narrow ovoids, though their features are coarse and the upper lips short. No. 193 and perhaps 336 might well have been introduced into fig. 46.

The components of fig. 45 make a good composite, but we are willing to allow that they do not possess typically "broad faces," nor the features generally called "strumous."

On the other hand, fig. 46 and its components have undoubtedly broad faces and powerful lower jaws, but they have not the broken noses nor short upper lips of the strumous face.

The features of the faces in fig. 47 are similar; all have the mouth open, a short upper lip, and a broad nose with more or less depressed bridge, yet the outlines of the faces are narrow rather than broad.

Fig. 48 and its components exhibit the broad face and characteristic features of struma when not sufficiently strong to be ill-favoured or deformed; they belong to much the same class of faces as fig. 31 among the women.

In closing this review and criticism of our own plates, we must express our great regret that some unforeseen pressure of

time at the last did not allow us to revise our male composites before publishing them; if we had been able to do so, we should have obtained better results. We dealt with the women first, and by submitting them to revision, we have procured more characteristic faces than at first. We would also draw attention to the fact that this is the first attempt at applying the new process of composite portraiture on a large scale, and that many technical difficulties, mechanical and others, could only gradually be overcome.

There is one advantage, however, in submitting the photographs of men in a somewhat imperfect condition; it affords an opportunity of demonstrating errors in selection, and gives examples of one of the advantages of composites as tests of accuracy of selection and grouping.

Finally, we may say that our results appear to lend no countenance to the belief that any special type of face predominates among phthisical patients, nor to the generally entertained opinion that the narrow, ovoid, or "tubercular" face is more common in phthisis than *among other diseases*. Whether it is more common than among the rest of the *healthy* population we cannot at present say.

It is true that taking both sexes together we find 14·3 per cent. of faces that may be classed as "narrow ovoids" and 9·3 per cent. that come under the head of "broad faces with coarse features," making in all 23·6 per cent. of our cases which may be grouped under one or other extreme departure in either direction from the normal average; but we doubt if this is more than would be found among the general population. Our results are therefore negative, but it may be they are no less valuable; although we commenced our investigations with the expectation of establishing a "type" on a firm foundation, we shall be little less satisfied with them if they have succeeded in refuting an error.

Although these conclusions would seem to indicate that there is no foundation for the belief that persons possessing certain physical characteristics are especially liable to tubercular disease, yet it may hereafter be proved that some explanation of the doctrine may be found in the course of the disease when it attacks such persons. In suggesting this we are going beyond the facts recorded in our present inquiry,

but the suggestion appears warranted by daily observation. Thus, the delicately organised individuals called "tubercular," and characterised by their "narrow ovoid" faces, have been compared with horses and cattle who have been what is called "over bred;" such animals are described as having too much nerve and too little bone and muscle; they have no "staying power" and readily "knock-up." In like manner these more delicately formed individuals, with highly susceptible nervous systems, well exemplified in the "precocious child," are little able to stand the strain and racket of disease, of whatsoever sort it may be, and more readily fall victims to its attacks than their more robustly built fellow-creatures.

Again, if it be true, as frequently asserted, that those having the features called "strumous" probably inherit a more or less diluted syphilitic taint; it is not surprising that they should be especially liable to inflammatory changes of a low type, and that disease in them should be readily amenable to treatment, especially by mercury, a result commonly seen in the so-called "strumous" diseases of children and often in those of adults.

These questions we hope to take up again hereafter, when possibly we may be able to demonstrate that though much error has been accumulated around the doctrine of "diatheses," it nevertheless contains a nucleus of valuable truth.

DESCRIPTION OF PLATES I, II, III, IV.

The *composites* are in medallions, the *original photographs* are in small squares. The composites are numbered consecutively as Figs. 1, 2, 3, &c.

The small numbers attached to the composites and to the photographs of individuals are for the purpose of identification in our indices of composites and of cases.

The faces are classified under the following heads; the references in the columns being to a good composite specimen of each variety:—

	Phthisical.		Non-phthisical.	
	Male.	Female.	Male.	Female.
General average	FIG. 17	FIG. 7	FIG. 11	FIG. 1
Narrow ovoid ("tubercular" type):				
<i>a.</i> Delicate and regular	36	29	12	2
<i>b.</i> Coarse and thickened	34	30		
Broad faces, thick features ("strumous" type):				
<i>a.</i> Somewhat comely	48	31		
<i>b.</i> Coarse and deformed	47	32		
Emaciated	22 or 18	10		

PLATE I

Contains *general averages* of phthisical and non-phthisical patients, both males and females (except Figs. 2, 10, and 12).

FIGS. 1 and 3.—Female non-phthisical patients (each contains 50).

FIG. 2.—Do., with narrow ovoid faces (contains 15). Compare also Figs. 29 and 30, Plate II.

FIGS. 7, 8, and 9.—Female phthisical patients, taken without selection (each contains 50).

FIG. 5.—Do., with strong hereditary taint (contains 56).

FIGS. 4 and 6.—Do., components of Fig. 5.

FIG. 10.—Do., with advanced disease (contains 11).

FIG. 11.—Male non-phthisical patients (contains 100).

FIGS. 13, 14, 15, and 16.—Do., components of Fig. 11 (each contains 25).

FIG. 12.—Do., with narrow ovoid faces (contains 13).

FIG. 17.—Male phthisical patients, taken without selection (contains 200).

FIGS. 18, 19, 20, and 21.—Do., components of Fig. 17 (50 in each).

PLATE II.

FIG. 22.—Male phthisical patients, with advanced disease (contains 42).

FIGS. 23, 24, 25, 26, 27, 28.—Do., do., components of Fig. 22 (each contains 7).

FIG. 29.—Female phthisical patients, narrow ovoids, high type (contains 9).

FIG. 30.—Do., narrow ovoids, low type (contains 12).

FIG. 31.—Do., broad faces, with comely features (contains 10).

FIG. 32.—Do., do., with deformed features (contains 5).

PLATE III.

FIG. 33.—Male phthisical patients, co-composite of all narrow ovoids (contains 51).

FIGS. 34, 35, 36, 37, 38, 39, 40.—Do., components of Fig. 33, with the individual portraits.

PLATE IV.

FIGS. 41, 42.—Male phthisical patients, components of Fig. 33 (continued from Plate III).

FIG. 43.—Do., co-composite of broad faces with thick features (contains 27).

FIGS. 44, 45, 46, 47, 48.—Do., components of Fig. 43, with the individual portraits.

Non phthisical cases



Fig. 1, to composite of 50



Fig. 2, to composite of 1000 words



Fig. 3, to composite of 500

Hereditary disease



Fig. 4, 8° 150



Fig. 5, 8° 150, of 50



Fig. 6, 8° 150

Phthisical cases



Fig. 7, 8° 150



Fig. 8, 8° 150



Fig. 9, 8° 150



Fig. 10, 8° 150

Phthisical disease

Non phthisical cases



Fig. 11, to composite

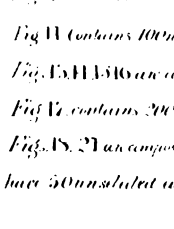


Fig. 12, to composite, 3000 words

Fig. 11 contains 100 non phthisical males

Fig. 13-15 are a composite, included in it.

Fig. 16 contains 200 unselcted cases of Phthisis

Fig. 18, 21 are composite, contained in Fig. 16, each have 50 unselcted cases of Phthisis.



Fig. 12, to composite, 3000 words



Fig. 13, 8° 150, 150



Fig. 14, 8° 150, 150



Fig. 15, 8° 150, 150



Fig. 16, 8° 150, 150

Phthisical cases



Fig. 17, to composite



Fig. 18, 8° 150



Fig. 19, 8° 200, 200



Fig. 20, 8° 200, 200



Fig. 21, 8° 200, 200

Thin neck case



Fig. 22. *Compensate*



Fig. 23. *N° 176*



Fig. 24. *N° 177*



Fig. 25. *N° 178*



Fig. 26. *N° 180*



Fig. 27. *N° 182*



Fig. 28. *N° 183*

Narrow oval face with features



Fig. 29. *N° 186*



Narrow oval face (low jaw)



Fig. 30. *N° 191*



Also see
Fig. 32

Broad face with thick upper lip (Struma)



Fig. 31. *N° 194*



Broad face (low jaw)



Fig. 32. *N° 195 (Struma, Syphilis?)*



Fig.
33.



Co-composites
of males having
narrow oval
faces



Fig. 34.



Fig.
35.



Fig.
36.



Fig.
37.



Fig.
38.



Fig.
39.



Fig.
40.



Components of "Narrow oval" Composites (Fig. 33)

Fig 41



Narrow snout

Fig 42



Fig 44

Fig 43



Composite Broad faces

Fig 45



Fig 47



Fig 48



Components of Broad and Thick "Composite (Fig 43)

ON

SOME POINTS IN RELATION TO INTRA-
OCULAR GLIOMA.

By W. A. BRAILEY, M.D.

THE precise origin of this form of tumour can only be inferred from its structure and from the relation of its spreading edge to the different retinal layers, since in all the cases that have hitherto come under examination these last are no longer recognisable in the body of the tumour.

Its cells are not unlike the retinal granules in structure and size. Their farthest extension into the adjacent retina may either be in the granules, inner or outer, or in the nerve fibre and cell layers. The molecular layers, the rods and cones, the fibres of Müller, and the elements of the pars ciliaris retinæ, are simply, one after the other, destroyed by pressure. And though the tumour cells multiply abundantly among the fibres of the optic nerve, yet its connective trabeculæ as well as the sheath of its central vessels remain free from them.

Glioma would thus appear to be a tumour derived from some form of nervous tissue, and not from the ordinary fibrous connective tissues. For though some of the bodies constituting the inner granules are undoubtedly the nuclei of the connective-tissue fibres of Müller, yet the great majority are, like the outer granules, undoubtedly nervous. In the same way,

though the molecular layers are traversed by fine nerve fibrils, yet the bulk of their tissue is made up of a fine connective-tissue reticulum derived from the Müllerian fibres. It is also reasonable to accept the idea that the tumour is due to a proliferation of the nuclei of the delicate structureless neuroglial basis-substance rather than to an increase of the functional nervous elements themselves.

The nutrient vessels of glioma, though abundant, are never large and thin walled, neither are the tumour cells directly in contact with their walls, but the two are always separated by a narrow zone of loose connective tissue, which constitutes a lymphatic sheath for each vessel. In both these respects glioma is different from the common form of intraocular sarcoma. Each capillary is surrounded by a zone of better nourished cells, so that the tumour, when it is not of very recent growth, is divided into a number of areas, the cells of which stain well and are well defined. The cells intermediate between the abruptly-defined borders of these are larger, less defined, and possess less staining capacity. It is here that the white gritty particles so characteristic of intraocular glioma are so commonly found.

By the rupture of the internal limiting membrane glioma cells spread as flocculent extensions into the vitreous chamber, or as a thin layer on the inner retinal surface, even covering the pars ciliaris retinae. This increase, which is due to continuous cell multiplication, is best marked in the form known as *G. endophytum* (Hirschberg). Extension into the choroid takes place in just the same manner, for it would appear that the endothelial cells of its lamellæ never take on the characters of glioma cells. Thus there is no diffuse hypernucleation of the choroid in advance of the solid mass or extending lines of glioma cells.

The opposite is the case in the optic nerve, for, though solid columns of cells are seen extending themselves in the position of the nerve fibre bundles, the nerve beyond their abrupt termination is uniformly hypernucleated. The change is almost entirely due to an increase in the number and size of the neuroglial corpuscles, which not only have acquired a resemblance to glioma cells, but appear to have gained some of their properties. In this way it is possible to explain the

fact that if, during enucleation, the nerve be cut at such a part, the tumour may still recur in its stump.

The pressure causes speedy degeneration of the tumour cells within the distended nerve, whereas those which occasionally occupy the intervaginal space keep their outlines much longer. The trabecular tissue and nerve sheaths even become hypertrophied. The lamina cribrosa appears to furnish the principal obstacle to the tumour cells; this once passed, their progress is more rapid.

Diagnosis.—Had the recognition of glioma been easy we should have known no such term as pseudo-glioma. But the difficulty or impossibility of ascertaining the degree of vision, its extent, the amount of pain, and even the tension, prevents the early recognition of even the existence of any morbid condition, and makes the diagnosis of its exact nature difficult.

The conditions which, during life, may be mistaken for glioma, depend principally upon changes in the vitreous cavity with or without detachment of retina. The most common is the formation of connective tissue, whose fibres, starting from the ciliary body, and to a less extent also from the adjacent posterior surface of the base of the iris, have firm adhesions to the posterior lens capsule and to the retina. By their contraction they detach the latter and draw back the iris periphery towards the circumlental space. On the other hand, the lens is thrust forwards, so that the centre of the aqueous chamber is shallow: the sclero-corneal junction is pulled in and the tension of the eye is diminished. A simple growth of connective tissue from the papilla may very rarely be mistaken for glioma. Detachment of retina with almost complete obliteration of the vitreous chamber may occur unnoticed in consequence of a spontaneous suppurative hyalitis. Far more rarely, a spontaneous diffuse uveitis with proliferation of the retinal pigment layer will produce the same results. Both these conditions may be included under pseudo-glioma, a term which is less or more comprehensive according to the degree of knowledge of the person employing it.

The diagnostic characters concern the appearances visible through the pupil, the tension, the condition of the anterior chamber, and, to a certain extent, the history. The light-coloured reflex of glioma shows many blood-vessels and frequent

hæmorrhagic spots. That of new-formed connective tissue or detached retina is comparatively free from them. In glioma the periphery of the anterior chamber is usually shallow, or, it may be, entirely obliterated. In pseudo-glioma the centre is shallow, while the peripheral part is abnormally deep. A consequent characteristic grooving of the iris near and parallel to its outer margin is very frequent. The tension, though normal in the earlier stages of glioma, is increased in the vast majority of cases observed. But the tension of pseudo-glioma is almost invariably subnormal. The value of this symptom, and also of the condition of the anterior chamber, is diminished by the occurrence among children of rare cases, where retinal detachment is accompanied by increased tension. Such a case is figured in Becker's Atlas, Part III, Plates 21 and 29. In those I have myself observed, the subretinal space was filled with blood and the characteristic glaucomatous changes of iris and ciliary body were present.

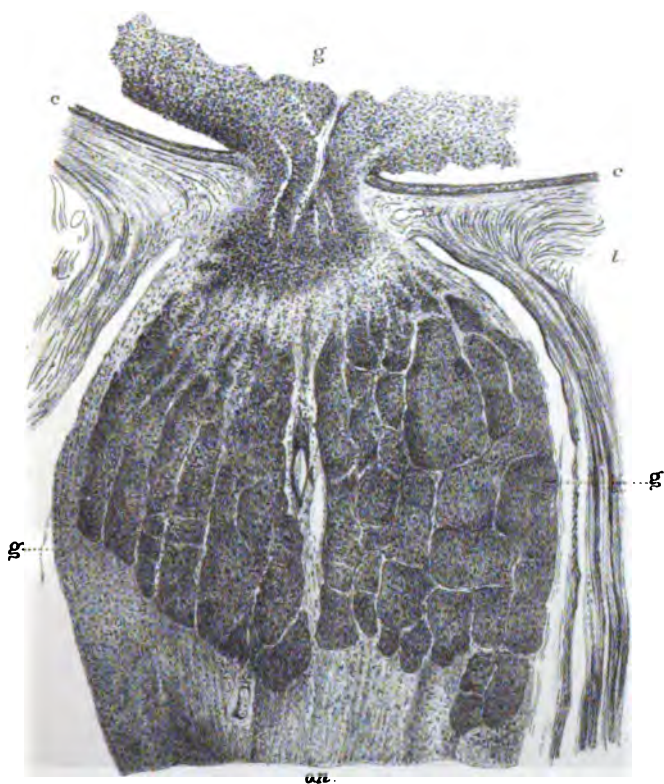
A previous febrile attack, especially if accompanied by cerebral symptoms, points towards pseudo-glioma. Head symptoms, when due to glioma, are late in their manifestation.

DESCRIPTION OF PLATES I AND II.

PLATE 1.—Glioma endophytum. Section through optic disc showing the granule layers uninvolved, whereas the nerve cells and fibres are replaced by tumour cells, which extend into the papilla, and also project as a solid mass into the vitreous cavity.

PLATE 2.—G. retinae. Showing the mode of extension into the optic nerve. The entire retina in the neighbourhood of the disc has disappeared in the tumour. The lamina cribrosa, though its anterior surface is very concave, is still recognisable. Columns of cells (cut, however, somewhat obliquely) extend along the enlarged nerve nearly as far as its divided end. Beyond this there is considerable hypernucleation of the nervous tissue. The trabeculae and the sheaths of the central vessels are uninvolved.





L I S T
OF
GENTLEMEN EDUCATED AT GUY'S HOSPITAL
WHO HAVE PASSED THE
EXAMINATIONS OF THE SEVERAL UNIVERSITIES, COLLEGES,
&c., &c.,
IN THE YEAR 1879.¹

University of Cambridge.

Final Examination for the Degree of Bachelor of Medicine.

E. C. Beale.

Second Examination for the Degree of Bachelor of Medicine.

F. A. Mahomed.

J. E. Viney.

J. Mackern.

First Examination for the Degree of Bachelor of Medicine.

F. A. Mahomed.

J. E. Viney.

University of London.

Examination for the Degree of Doctor of Medicine.

R. E. Carrington.

G. B. Raine.

L. H. Stevenson.

J. O. Uhthoff.

Logic and Psychology only.

A. Buchanan.

M. Lubbock.

¹ The Editors will thank past and present Students of Guy's Hospital for any information as to Degrees, Diplomas, or Honours obtained by them during the current year (1881).

502 *Gentlemen admitted to Degrees, &c., in the year 1879.*

Examination for the Degree of Bachelor of Medicine.

First Division.

T. W. Fuller.

Obtained the Gold Medal in Obstetric Medicine, and Honours in Medicine.

G. Mackern.

Obtained First-Class Honours with the number of Marks qualifying for a Gold Medal in Medicine.

T. W. O. Pughe.

Obtained First-Class Honours in Medicine, and Honours in Forensic Medicine and in Obstetric Medicine.

W. H. White.

Obtained Honours in Medicine.

G. H. Russell.

Second Division.

A. E. Maylard.

Obtained Honours in Forensic Medicine.

Examination for the Degree of Bachelor of Surgery.

First Division.

A. E. Maylard.

Second Division.

T. Jones.

Obtained Honours in Surgery.

Intermediate Examination in Medicine.

First Division.

E. L. Adeney.

Obtained the Exhibition and Gold Medal in Organic Chemistry.

W. E. Fielden.

Obtained First-Class Honours in Histology and Physiology, and Honours in Materia Medica and Pharmaceutical Chemistry.

W. A. Lane

Obtained Gold Medal in Anatomy, and number of Marks qualifying for Exhibition, Honours in Histology and Physiology, and First-Class Honours with number of Marks qualifying for Medal in Organic Chemistry.

Second Division.

R. Prothero.

Obtained Honours in Organic Chemistry.

J. J. Udale.

Obtained Honours in Organic Chemistry and in Anatomy.

F. T. Bayes.

J. I. Boswell.

O. J. Currie.

L. E. Shaw.

Excluding Physiology.

R. A. Milligan.

Physiology only.

J. Smith.

Preliminary Scientific (M.B.) Examination.

First Division.

R. A. Bindley.
H. C. E. Cooper.
H. C. Ensor.
W. W. Floyer.
A. Green.
W. Hind.

A. Martin.
E. O. Newland.
F. W. H. Penfold.
W. A. Slater.
C. S. Spong.
J. H. Targett.

T. B. Winter.

Second Division.

C. E. Player.

W. D. Smallpeice.

Examination for the Degree of Bachelor of Science.

J. G. Ridsdale.

Obtained Honours in Botany.

University of Durham.

Final Examination for the Degree of Bachelor of Medicine.

W. J. Tyson.

University of Edinburgh.

Final Examination for the Degree of Bachelor of Medicine.

J. M. Hobson.

Second Examination for the Degree of Bachelor of Medicine.

J. F. Codner.
F. O. Combe.
J. M. Hobson.

O. S. Magrath.
G. C. S. Perkins.
J. F. Tabb.

First Examination for the Degree of Bachelor of Medicine.

J. F. Codner.

F. O. Combe.

University of Aberdeen.

Examination for the Degree of Doctor of Medicine.

H. Bartlett.

Final Examination for the Degree of Bachelor of Medicine.

W. Brown.

E. Field.

A. H. Burton, B.A.

W. Stericker.

F. J. Fehrsen.

Second Examination for the Degree of Bachelor of Medicine.

H. J. Liebshtein.

J. W. Hodgson.

Examination for the Degree of Master in Surgery.

A. H. Burton, B.A.

E. Field.

F. J. Fehrsen.

W. Stericker.

Queen's University in Ireland.

Second Examination for the Degree of Doctor of Medicine.

J. M. Prendergast.

Indian Medical Service.

London Examination in August.

H. O. Stuart, 2083 marks.

A. G. Collington, 1860 marks.

Netley Examination in February.

C. G. W. Lowdell, 4264 marks.

Army Medical Service.

London Examination in December.

A. H. Keogh, 2525 marks.

H. E. B. Flanagan, 2295 marks.

J. G. S. Lewis, 2065 marks.

R. O. Cusack, 2010 marks.

F. E. C. Hood, 1625 marks.

Naval Medical Service.

Netley Examination in February.

A. M. French, 3507 marks.

E. H. Williams, 3504 marks.

Royal College of Physicians, London.

Examination for the Membership.

R. E. Carrington, M.D. | D. W. O. Hood, M.D.

Examination for the Licence.

T. A. Bell.	T. A. I. Howell.
J. Osborne.	J. Poland.
J. T. Gardner.	P. M. Wood.
J. G. S. Lewis.	H. L. Bates.
A. H. Pierson.	W. T. Crew.
R. W. White.	G. S. Lewis.

Sydney Smith.

Royal College of Surgeons.

First Examination for the Fellowship.

E. Worts. | J. W. Sanders. | D. C. Trott.

Final Examination for the Membership.

January.

F. F. Jones.	H. Dismorr.	R. W. White.
G. H. Russell.	E. J. Morley.	T. W. Fuller.
A. E. Cheshire.	E. Roper.	J. T. J. Morrison.
J. W. Collington.		

April.

G. F. Crooke.	J. F. Tabb.	A. H. Pierson.
E. A. Jones.	J. G. Gravely.	R. J. H. Scott.
W. J. Coles.	A. K. Morgan.	R. T. Bedford.

May.

F. T. Logan.	E. Penny.	C. A. Everest.
F. B. J. Baldwin.	J. G. Barns.	T. A. Bell.
H. Hine.	R. Parry.	J. Poland.

July.

J. G. S. Lewis.	J. S. Crook.	F. J. Elliott.
H. E. B. Flanagan.	R. E. R. Morse.	E. O. Jago.
W. T. Crew.	J. Smith.	A. Scott.
A. D. Deane.	H. E. Walker.	F. T. Wilkinson.
H. A. Clowes.	H. E. Wright.	P. St. G. Williams.

November.

C. H. Haycroft.	R. Steele.	B. N. Rake.
T. A. I. Howell.	C. St. John Wright.	J. R. Rolston.
H. J. Liebstein.	G. H. W. Jones.	L. C. Wooldridge.
G. S. Pilkington.	John Cock.	C. J. Plummer.
Sydney Smith.	T. H. Morse.	J. W. Sanders.

506 *Gentlemen admitted to Practice, &c., in the year 1879.*

First Examination for the Membership.

January.

T. A. I. Shepherd.	C. C. Brodrick.	S. A. Davies.
H. G. Stacy.	T. M. Day.	S. T. M. Evans.
J. C. Pincott.	F. V. Duckworth.	A. T. Perkins.
H. B. Hawksworth.	J. E. Anderton.	A. Bolton.
T. B. Luscombe.	L. Stokes.	G. W. Mullis.
G. F. P. Pizey.	W. Spong.	G. F. Dixon.
B. B. A. Taylor.	J. F. Spong.	

April.

B. P. Bartlett.	J. H. Champ.	E. L. Adeney.
W. H. W. Strachan.	H. M. Bayliss.	E. S. Dashwood.
W. C. Dendy.	P. Warner.	L. E. Shaw.
W. E. Starling.	B. Scott.	P. Pigott.
E. H. Booth.	J. Dowson.	Z. Prentice.
G. N. Pitt.	J. A. P. Price.	

May.

T. Unicum.	F. M. Pedley.	G. L. L. Lawson.
W. H. Hart.	F. E. Row.	J. B. Trapp.
G. S. Mahomed.	F. E. Hubbard.	E. Aphthorp.
H. Blatherwick.	C. R. O. Garrard.	J. J. Faraker.
L. E. W. Stephens.	A. G. Wildey.	C. F. Campe.
F. Eastes.	H. A. Fotherby.	J. W. F. Long.
G. Utting.	G. S. Pollard.	G. H. Capes.
G. T. Woolley.	H. R. Osborne.	G. J. Currah.
R. H. Perks.	H. C. Dixon.	A. J. Hind.
W. S. N. Shorthouse.	H. E. Rowell.	M. O'Kane.
S. O. Stuart.	F. C. Payne.	W. E. Rudd.
J. J. Palmer.		

July.

C. T. Griffiths.	E. Roberts.
T. F. W. Rowlands.	J. J. Tomney.
C. S. Harper.	

November.

J. A. Fraser.	A. H. Jackson.
H. W. Moor.	

Apothecaries' Society.

Final Examination for the Licence.

R. Parry.	W. P. Bothamley.
H. E. B. Flanagan.	E. N. Davies.
G. F. Orooke.	C. St. John Wright.
J. Osborne.	N. E. Johnson-Gaylor.
J. F. Tabb.	R. T. Jones.
J. T. Gardiner.	G. E. Pollard.
C. H. Downes.	W. F. Hearnden.
E. Penny.	W. T. Crew.
M. D'O. Gilkes.	C. H. Haycroft.
J. G. S. Lewis.	H. J. Liebenstein.
J. C. Uthoff.	H. E. Wright.
F. Hitch.	

First Examination for the Licence.

N. E. Johnson-Gaylor.	B. R. A. Taylor.
T. E. Abbott.	Z. Prentice.
B. Studer.	T. R. Atkinson.
C. H. Downes.	G. S. Mahomed.
J. G. S. Lewis.	T. A. J. Shepherd.
T. B. Luscombe.	J. B. Trapp.
R. A. H. Hart.	S. O. Stuart.
H. R. Osborne.	B. P. Bartlett.
W. H. Crosse.	A. W. Moore.
G. J. Currah.	C. F. Campe.

University of London.

Matriculation Examination.

January.

E. H. Armitage.	W. Hind.
H. C. E. Cooper.	A. Martin.
J. Curtis.	J. H. Targett.
W. D. Smallpeice.	

June.

L. A. Dunn.	C. H. L. Meyer.
R. Lawson.	G. M. R. Pollard.
W. M. Woodhouse.	

MEDALLISTS AND PRIZEMEN, 1878-79.

JULY, 1879.

The Treasurer's Gold Medal for Medicine.

Harold Edward Bickerson Flanagan, Woolwich.

The Treasurer's Gold Medal for Surgery.

James Thomas Jackman Morrison, Plumstead.

Third Year's Students.

George Ryding Marsh, London
 John William Sanders, Haverfordwest } (equal) £27 10s.
 Edwin A. Starling, Sutton, Certificate.

Second Year's Students.

JOSEPH HOARE PRIZES.

Edwin Leonard Adeney, Reigate, First Prize, £25.
 John Alfred Parry Price, Brecon, Second Prize, £10.
 Edward Hargrave Booth, London, Certificate.
 Walter Chester Dendy, Brighton, Certificate.
 Robert Howell Perks, Cardiff, Certificate.
 Lockhart E. W. Stephens, Emsworth, Certificate.

MICHAEL HARRIS PRIZE.

J. A. Parry Price, Brecon.

First Year's Students.

Edwin James Wenyon, Darlington, First Prize, £50.
 Louis Albert Dunn, Second Prize, £25.
 Walter Thomas Harris, London, Third Prize, £10 10s. •
 Sydney Worthington, Liverpool, Certificate.
 Holland Hodgson Wright, Forest Hill, Certificate.
 Alfred E. C. Woodhouse, Manchester, Certificate.
 William T. F. Davies, Swansea, Certificate.

SEPTEMBER, 1879.

Open Scholarship in Arts.

George Ezra Halstead, Newark.

Open Scholarship in Science.

Walter Fowler, London.

OCTOBER, 1879.

Gurney Hoare Prize.

Beaven Neave Rake.

Pupils' Physical Society.

Session 1879-80.

Honorary President.—Dr. WILKS.

Presidents.

Messrs. F. J. Bayes, J. Cock, C. H. Downes, H. E. B. Flanagan,
 T. H. Morse, G. Mackern, A. E. Maylard, J. T. J. Morrison, E. Penny,
 B. N. Rake, G. H. Russell, A. Scott, O. B. Shelswell, H. Sturge,
 W. E. Starling, W. H. White, P. M. Wood, and L. C. Wooldridge, B.Sc.

Honorary Secretaries.—R. CLEMENT LUCAS, B.S.; C. J. SYMONDS, B.S.

PRIZEMEN FOR THE SESSION 1878-79.

To Mr. C. H. Downes, £10, for his Paper on "Acute Rheumatism and Salicylic Acid."

To Mr. H. H. Sturge, £5, for his Paper entitled "Notes on Skin Disease."

To Mr. R. Parry, £5, for his Paper on "Rest and Position in the Treatment of Medical Cases."

To Mr. W. E. Starling, £5, for his Essay on "The Physiology and Histology of the Lymphatic System."

CLINICAL APPOINTMENTS HELD IN THE YEAR 1879.

RESIDENT HOUSE PHYSICIANS.

G. Mackern, M.B.	E. H. Paddison, M.B.
J. C. Uhthoff, M.B.	G. F. Crooke, M.B.
H. Davy, M.B.	F. B. J. Baldwin.

RESIDENT HOUSE SURGEONS.

C. K. Shaw.	C. H. Keep.
R. S. Wainewright, M.B.	F. F. Jones.
J. W. Meek, M.B.	E. Penny, M.B.

RESIDENT OBSTETRIC ASSISTANTS.

E. B. Granger.	G. H. West Jones.	F. F. Jones.
E. J. Morley.	E. Penny, M.B.	T. F. Pedley.
C. Wood.	J. Poland.	R. W. White.
J. Osborne.	E. C. Beale.	H. E. B. Flanagan.
E. Roper.		

SURGEONS' DRESSERS.

D. D. Malpas.	H. H. Austin.	F. T. Wilkinson.
T. A. I. Howell.	A. K. Morgan.	T. H. Morse.
J. Alexander.	T. B. Cross.	L. C. Wooldridge.
J. F. Briscoe.	A. Scott.	W. T. Crew.
H. L. Bates.	E. S. Cockell.	J. S. Crook.
W. H. White.	G. C. S. Perkins.	W. Whitworth.
R. R. W. Oram.	H. E. B. Flanagan.	H. H. Bovill.
F. J. Elliott.	J. T. J. Morrison.	B. N. Rake.
F. F. Jones.	P. M. Wood.	

CLINICAL ASSISTANTS.

A. E. Maylard, M.B., B.S.	T. W. Fuller, M.B.	R. F. Cox.
G. H. W. Jones.	W. H. White, M.B.	T. H. Morse.
R. Parry.	G. Pilkington.	E. A. Starling.
R. W. White.	R. Steele.	A. Scott.
H. E. Wright.	H. H. Sturge.	F. S. Pilkington.
P. M. Wood.	P. St. G. Williams.	F. T. Wilkinson.

DRESSERS IN THE EYE WARDS.

A. G. Barra, M.B.	C. Atkin.	H. L. Bates.
F. H. Berry, M.B.	J. S. Crook.	T. A. I. Howell.
C. H. Parke.	E. B. Granger.	C. T. Harper.
T. W. Fuller, M.B.	A. E. Maylard, M.B., B.S.	H. H. Austin.
M. J. Hart.	R. W. White.	C. H. Downes.
E. Penny, M.B.	J. F. Briscoe.	G. J. Wilson.

DENTAL SURGEON'S DRESSERS.

J. F. Tabb.	A. T. Perkins.	G. F. Dixon.
D. C. Trott.	G. J. Cressy.	F. O. Payne.
J. R. Rolston.	W. H. Crosse.	W. D. J. Morris.
F. H. Shanks.	F. E. Row.	

MEDICAL CLINICAL CLERKS.

C. J. Plummer.	W. T. Crew.	W. W. Pryn.
W. J. Parkinson.	F. O. Combe.	J. Rigby.
J. F. Tabb.	E. R. D. Fasken.	H. P. Rowlands.
R. J. H. Scott.	M. D'O. Gilkes.	B. Studer.
J. T. Brett.	A. H. Langridge.	F. H. Shaw.
W. P. Bothamley.	F. W. Pilkington.	E. G. Williams.
H. A. Clowes.	E. A. Starling.	H. E. Charles.
J. S. Crook.	G. J. Wilson.	J. Smith.
C. H. Downes.	H. E. Archer.	H. Hawksworth.
E. O. Jago.	H. W. Campbell.	T. B. Luscombe.
T. H. Morse.	G. R. Marsh.	L. W. K. Phillips.
G. O. S. Perkins.	H. G. Ashwell.	J. M. Prendergast.
F. S. Pilkington.	L. Burroughs.	J. R. Rolston.
H. A. Phillips.	C. Clay.	D. C. Trott.
J. A. Smith.	H. D. Davenport.	H. T. Bassett.
S. T. Thomas.	W. W. David.	H. A. Fotherby.
J. Anderson.	S. A. Davies.	J. W. Woodruff.
C. H. Haycroft.	E. Elliott.	T. R. Atkinson.
A. P. Hills.	P. H. Gardner.	W. O. Fenwick.
R. T. Jones.	A. L. Lane.	W. O. Hearnden.
F. M. Godde-Smith.	A. C. Otway.	C. T. Griffith.

ASSISTANT-SURGEON'S DRESSERS.

A. H. Langridge.	P. H. Gardner.	D. C. Trott.
H. E. Archer.	J. W. Woodruff.	T. F. Bayes.
G. R. Marsh.	T. R. Atkinson.	J. W. Hodgson.
H. P. Rowlands.	H. Charles.	G. F. Dixon.
H. T. Bassett.	W. C. Hearnden.	F. Eastes.
A. C. Otway.	B. H. Lane.	A. P. Hills.
A. L. Lane.	A. T. Perkins.	F. M. G. Smith.
F. Hitch.	A. J. Hind.	W. T. Crew.
S. T. M. Evans.	E. G. Williams.	G. J. Wilson.
J. M. Owen.	G. H. Oapes.	S. A. Davies.
J. W. Sanders.	E. R. D. Fasken.	B. N. Rake.
W. Sponge.	F. W. Pilkington.	L. Burroughs.
J. Harrison.	J. R. Rolston.	F. H. Shaw.
R. T. Jones.	W. W. Pryn.	T. M. Day.
D. G. Edwards.	T. B. Luscombe.	W. O. Fenwick.
G. W. Mullis.	A. S. Stokes.	M. D'O. Gilkes.
J. Anderson.	B. Studer.	W. H. O. Newnham.
John Smith.	J. Rigby.	J. W. Nicholson.
L. W. K. Phillips.	N. E. Johnson-Gaylor.	B. Scott.
J. M. Prendergast.	J. C. Pincott.	H. M. Bayliss.
E. Elliott.	L. Stokes.	G. T. Woolley.
C. Clay.		

DRESSERS IN THE SURGERY.

H. Charles.	W. O. Hearnden.	H. E. Rowell.
J. H. Harris.	G. W. Mullis.	B. Scott.
F. T. Bayes.	A. T. Perkins.	S. O. Stuart.
A. Hind.	J. C. Pincott.	P. Warner.
H. W. Campbell.	G. F. P. Pizey.	D. G. Edwards.
F. O. Combe.	W. E. Starling.	B. P. Bartlett.
M. D'O. Gilkes.	T. A. J. Shepherd.	W. Dendy.
B. N. Rake.	L. Stokes.	J. Dowson.
G. J. Cressy.	J. F. Spong.	E. D. Minter.
H. T. Bassett.	W. Spong.	W. D. J. Morris.
E. Elliott.	W. H. C. Newnham.	G. N. Pitt.
H. Gard.	H. M. Bayliss.	F. E. Row.
N. E. Johnson-Gaylor.	A. Bolton.	H. R. Osborne.
A. L. Lane.	H. Blatherwick.	F. N. Pedley.
A. S. Stokes.	F. V. Duckworth.	G. S. Pollard.
J. W. Woodruff.	G. L. L. Lawson.	W. E. Rudd.
J. Rigby.	J. Harrison.	T. Unicume.
S. Edwards.	J. W. Long.	L. E. Shaw.
S. T. M. Evans.	B. R. A. Taylor.	E. H. Booth.
J. E. Anderton.	F. Eastes.	C. T. Griffiths.
C. C. Broderick.	H. A. Fotherby.	J. J. Tomney.
T. M. Day.	G. S. Mahomed.	A. G. Wildey.
G. F. Dixon.	J. J. Palmer.	W. E. Fielden.
H. Hawksworth.	Z. Prentice.	

AURAL SURGEON'S DRESSERS.

E. Fielding.	H. H. Sturge.	F. H. Berry.
P. St. G. Williams.	B. N. Rake.	A. T. Perkins.
S. H. Moore.	F. H. Shanks.	G. N. Pitt.

ASSISTANT-PHYSICIANS' CLERKS.

J. Cock.	S. T. Thomas.	F. H. Shaw.
W. H. Crosse.	R. T. Bedford.	H. G. Ashwell.
E. Elliott.	W. W. David.	R. Warner.
B. Studer.	F. J. Elliott.	S. H. Moore.
H. E. Archer.	L. W. K. Phillips.	B. N. Rake.
L. W. K. Phillips.	C. J. Harper.	H. W. Gosse.
H. E. Charles.	O. B. Shelswell.	J. S. Smith.
John Smith.	J. W. Sanders.	E. Penny.
E. Roper.	C. Sage.	J. R. Rolston.
W. A. Phillips.	H. W. Campbell.	G. F. Dixon.
W. P. Bothamley.	A. P. Hills.	

POST-MORTEM CLERKS.

P. Pigott.	H. G. Ashwell.	T. R. Atkinson.
J. J. Udale.	H. H. Bovill.	E. S. Cockell.
F. N. Pedley.	H. Charles.	H. W. Gosse.
C. Clay.	E. A. Starling.	W. E. Rudd.
P. H. Gardner.	W. H. Crosse.	F. E. Row.
H. A. Clowes.	W. W. Pryn.	

OBSTETRIC OUT-PATIENT CLERKS.

H. H. Sturge.	R. J. H. Scott.	F. V. Duckworth.
N.E.Johnson-Gaylor.	T. A. I. Howell.	A. S. Stokes.
J. W. Sanders.	H. E. Walker.	H. G. Ashwell.
B. Studer.	H. P. Rowlands.	F. H. Shaw.
R. T. Jones.	H. A. Clowes.	Wm. Spong.
B. N. Rake.	T. B. Luscombe.	G. O. S. Perkins.
W. A. Phillipps.	A. G. Wildey.	W. H. C. Newnham.
O. B. Shelswell.	H. E. Archer.	J. J. Udale.

EXTERNE OBSTETRIC ATTENDANTS.

C. Atkin.	F. S. Pilkington.	J. Anderson.
C. Clay.	S. T. Thomas.	O. J. Fookes.
F. O. Combe.	G. S. Mahomed.	F. W. Pilkington.
E. Elliott.	A. Bolton.	L. Stokes.
P. H. Gardiner.	H. Hawksworth.	G. J. Wilson.
F. Hitch.	T. Unicume.	T. M. Day.
H. Ashwell.	H. T. Bassett.	F. V. Duckworth.
T. W. Mullis.	T. B. Luscombe.	W. H. C. Newnham.
J. C. Pincott.	G. F. P. Pizey.	J. Rigby.
E. A. Starling.	J. W. Woodruff.	W. Spong.
F. H. Shaw.	L. C. Wooldridge.	E. L. Adeney.
W. O. Fenwick.	J. E. Anderton.	H. O. Dixon.
T. H. Morse.	T. A. J. Shepherd.	G. S. Pollard.
J. R. Rolston.	J. F. Spong.	J. P. Trapp.
J. Smith.	H. A. B. Davies.	J. J. Udale.
A. Lane.	T. B. Jacobson.	R. A. Milligan.
O. C. Broderick.	J. H. Booth.	J. W. Nicholson.
H. W. Gosse.	C. R. O. Garrard.	D. C. Trott.
N.E.Johnson-Gaylor.	A. J. Hind.	G. T. Woolley.

SURGICAL WARD CLERKS.

H. D. Davenport.	G. F. P. Pizey.	W. S. N. Shorthouse.
L. Burroughs.	L. Stokes.	T. Unicume.
W. O. Hearnden.	W. Spong.	G. T. Woolley.
A. L. Lane.	J. F. Spong.	G. N. Pitt.
A. S. Stokes.	B. R. A. Taylor.	A. Bolton.
B. Studer.	E. G. Hunt.	G. L. L. Lawson.
J. H. H. Williams.	G. E. Stamper.	P. Pigott.
H. T. Bassett.	D. E. Hickman.	O. J. Currie.
W. H. C. Newnham.	A. J. Hind.	J. H. Lister.
R. H. Browne.	C. O. Broderick.	J. W. F. Long.
D. G. Edwards.	E. S. Dashwood.	G. Utting.
H. P. Keatinge.	J. J. Faraker.	R. A. Milligan.
E. G. Williams.	B. P. Bartlett.	H. Blatherwick.
J. E. Anderton.	W. C. Dendy.	J. H. Champ.
R. Cuff.	J. Dowson.	G. S. Mahomed.
T. M. Day.	H. E. Dixon.	R. H. Perks.
S. T. M. Evans.	J. A. P. Price.	L. E. W. Stephens.
F. V. Duckworth.	F. N. Pedley.	P. Warner.
A. T. Perkins.	G. S. Pollard.	J. J. Palmer.
J. O. Pincott.	W. H. W. Strachan.	F. C. Payne.

ASSISTANT-SURGEONS' CLERKS.

F. E. Row.	W. J. C. Tomalin.	R. A. Milligan.
F. Pearse.	G. W. B. Slader.	E. T. Trevor.
J. O. Littlewood.	J. B. Trapp.	W. T. F. Davies.
C. Bishop.	W. E. Rudd.	A. M. Sutton.
D. T. Edmunds.	A. G. Wildey.	S. Worthington.
F. E. Hubbard.	E. R. B. Archer.	A. Scott, jun.
R. W. Brogden.	C. Y. Shuter.	H. P. Berry.
T. Unicum.	H. C. E. Cooper.	E. R. S. Lipscomb.
C. W. Bowles.	F. R. B. Bishopp.	A. Searle.
F. H. Shanks.	C. R. O. Garrard.	F. F. Jeyes.
E. S. Tresidder.	J. W. F. Long.	W. L. W. Marshall.

L I S T
OF
GENTLEMEN EDUCATED AT GUY'S HOSPITAL
WHO HAVE PASSED THE
EXAMINATIONS OF THE SEVERAL UNIVERSITIES, COLLEGES,
&c., &c.,
IN THE YEAR 1880.

University of Oxford.

First Examination for the Degree of Bachelor of Medicine.

J. A. P. Price.		R. J. Ryle.
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University of Cambridge.

Final Examination for the Degree of Bachelor of Medicine.

F. A. Mahomed.		J. Mackern.
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Second Examination for the Degree of Bachelor of Medicine.

G. N. Pitt.		W. Fowler.
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First Examination for the Degree of Bachelor of Medicine.

H. J. Hilbers.

University of London.

Examination for the Degree of Doctor of Medicine.

G. Mackern.		W. H. White.
M. Lubbock.		A. H. Jones.
A. Buchanan.		J. C. Ferrier.

Examination for the Degree of Master in Surgery.

O. J. Symonds.

Examination for the Degree of Bachelor of Medicine.

First Division.

J. W. Meek.

*Obtained the Scholarship and Gold Medal in Forensic Medicine, Honours in
Medicine and Obstetric Medicine.*

Gentlemen admitted to Degrees, &c., in the year 1880. 515

F. H. Berry.

Obtained First-Class Honours in Forensic Medicine.

E. Penny.

Obtained Honours in Medicine and Forensic Medicine.

R. S. Wainewright.

Obtained Honours in Medicine and Forensic Medicine.

Second Division.

H. A. G. Brooke.

Obtained Honours in Medicine and Forensic Medicine.

Intermediate Examination in Medicine.

First Division.

J. A. P. Price.

Obtained the Exhibition and Gold Medal in Physiology and Histology, and the Exhibition and Gold Medal in Chemistry, and Honours in Materia Medica.

L. E. W. Stephens.

Obtained Honours in Chemistry.

E. H. Booth.

Obtained Honours in Physiology and Histology.

R. H. Spicer.

Obtained Honours in Chemistry.

H. P. Berry.

|
S. Worthington.

J. H. Lister.

Second Division.

W. T. F. Davies.

|

J. W. Nicholson.

Excluding Physiology.

Robert Parry.

Preliminary Scientific (M.B.) Examination.

First Division.

G. E. C. Anderson.

Obtained Honours in Chemistry.

A. H. Fowler.

Obtained Honours in Chemistry.

H. W. Hart.

Obtained Honours in Chemistry.

W. A. Aikin.

L. A. Dunn.

|
W. I. Watson.

G. E. Halstead.

H. E. Jones.

Second Division.

L. F. Childe.

J. M. France.

C. H. Meyer.

|

O. D. Muspratt.

G. R. M. Pollard.

P. Parnell.

516 *Gentlemen admitted to Practice, &c., in the year 1880.*

Examination for the Degree of Bachelor of Science.

First Division.

W. A. Slater.

|

C. S. Spong.

University of Durham.

First Examination for the Degree of Bachelor of Medicine.

H. A. Clowes.

|

F. Eastes.

W. E. Rudd.

University of Edinburgh.

Final Examination for the Degree of Bachelor of Medicine.

F. O. Combe.

|

G. F. Crooke.

R. W. Lethbridge.

Final Examination for the Degree of Master in Surgery.

F. O. Combe.

|

R. W. Lethbridge.

*Final Examination for the Degree of Bachelor of Science in the
Department of Public Health.*

J. F. J. Sykes.

University of Aberdeen.

Examination for the Degree of Doctor of Medicine.

A. D. Brenchley.

|

L. Rudd.

Final Examination for the Degree of Bachelor of Medicine.

H. J. Liebshtein.

|

C. St. J. Wright.

Second Examination for the Degree of Bachelor of Medicine.

H. A. Phillips.

Examination for the Degree of Master in Surgery.

H. J. Liebshtein.

|

C. St. J. Wright.

Indian Medical Service.

London Examination in August.

A. G. Collington, 3755 marks.

R. E. R. Morse, 1600 marks.

|

T. C. Nugent, 1620 marks.

A. H. Pierson, 1790 marks.

Royal College of Physicians.

Examination for the Membership.

M. Lubbock, M.D.

Final Examination for the Licence.

H. H. Bovill.	C. Atkin.
H. Hine.	F. S. Pilkington.
B. N. Rake.	G. C. S. Perkins.
C. J. Plummer.	J. W. Sanders.

Royal College of Surgeons.

Final Examination for the Fellowship.

C. F. Pickering.

First Examination for the Fellowship.

J. A. P. Price.	E. H. Booth.	H. Blatherwick.
R. H. Perks.	F. Eastes.	W. C. Dendy.
W. S. N. Shorthouse.		

Final Examination for the Membership.

January.

C. Atkin.	C. E. Harper.	H. L. Bates.
W. P. Bothamley.	S. R. H. Mathews.	W. H. White.
W. W. David.	L. W. K. Phillips.	S. V. Theed.
M. D'O. Gilkes.	J. J. Reynolds.	W. Whitworth.
F. M. Godde-Smith.	J. S. Smith.	G. S. Lewis.

April.

H. P. Rowlands.	G. C. S. Perkins.	W. N. Puddicombe.
H. G. Ashwell.	H. H. Bovill.	J. R. Harris.
A. H. Langridge.		

May.

H. H. Sturge.	F. W. Pilkington.	J. Alexander.
H. T. Bassett.	F. Hitch.	T. B. Cross.
C. J. Plummer.	A. Smart.	F. T. Wilkinson.

July.

W. H. O. Newnham.	G. F. P. Pizey.	R. Bredin.
J. F. Briscoe.	G. J. Wilson.	S. H. Moore.
A. L. Lane.	A. P. Hills.	C. J. Parke.

November.

A. C. Otway.	T. M. Day.	H. W. Gosse.
W. W. Pryn.	E. S. Cockell.	D. O. Trott.
H. W. Campbell.	E. Fielding.	J. Smith.
F. T. Bayes.		

First Examination for the Membership.

January.

T. A. Evans.	W. Growse.	W. D. J. Morris.
Q. R. Veitch.	T. F. Jeyes.	J. B. Howell.
H. E. Richardson.		

518 *Gentlemen admitted to Degrees, &c., in the year 1880.*

April and May.

W. Hind.	M. Carnelley.	E. R. S. Lipscomb.
E. J. Wenyon.	R. A. Baillie.	W. H. Moore.
L. A. Dunn.	J. H. Cox.	J. O. Littlewood.
C. H. Meyer.	W. R. Etches.	A. J. Dalton.
J. H. H. Williams.	W. T. Harris.	L. McE. Anderson.
J. H. Booth.	J. B. Berry.	H. A. B. Davies.
G. Kendall.	W. E. Audland.	H. C. Ensor.
G. H. Graham.	H. H. Wright.	F. A. A. Bush.
M. Parry Jones.	W. C. Spiller.	D. T. Edmunds.
St. J. O. Rands.	R. J. Ryle.	J. G. Milnes.
A. E. O. Woodhouse.	H. T. Sells.	F. N. Shillingford.
A. Searle.	R. P. Samut.	G. H. Kinch.
S. Worthington.	J. V. Salvage.	H. B. Todd.
A. De Winton.	J. H. Targett.	H. G. Plimmer.
Thos. Carr.	E. G. Hunt.	C. S. Shuter.
A. T. F. Brown.	G. P. Longman.	J. M. Griffin.
W. T. F. Davies.	J. H. Gibson.	E. T. Trevor.
R. W. Brogden.	H. P. Keatinge.	J. C. Underwood.
C. E. Beebe.	W. H. Tomalin.	M. A. Muirhead.
B. M. Moorhouse.	G. R. Green.	

July.

A. S. Topham.	S. B. A. Edsall.	W. Wilson.
E. S. Tresidder.	J. J. D. Vernon.	A. M. Sutton.
T. Cardwell.	A. Green.	J. J. Prendergast.
W. D. Smallpeice.	J. H. Greenway.	L. Powell.
J. F. Saunders.	F. Chittenden.	A. Tireman.
E. O. Newland.	E. W. Roberts.	

November.

W. W. Floyer.	R. H. Browne.	A. P. H. Griffiths.
G. W. B. Slader.	J. A. Marsden.	R. L. Knaggs.
H. C. E. Cooper.		

Apothecaries' Society.

Final Examination for the Licence.

E. O. Jago.	W. H. Crosse.	W. W. Pryn.
B. Studer.	G. S. Mahomed.	H. H. Sturge.
C. J. C. Otway.	H. A. Fotherby.	F. P. Flood.
T. G. Beckett.	J. Smith.	H. R. Osborne.
C. J. Parke.	Alex. Lane.	H. W. Campbell.
R. Prothero.	G. C. S. Perkins.	G. J. Wilson.
C. F. Campe.	C. R. O. Garrard.	

First Examination for the Licence.

W. N. Puddicombe.	W. T. Harris.	J. J. Faraker.
H. A. Fotherby.	T. A. J. Shepherd.	E. S. Cockell.
C. R. O. Garrard.	G. J. Currah.	J. H. Harris.
W. C. Fenwick.	G. F. Symons.	L. W. K. Phillips.
C. J. Parke.	Alex. Lane.	H. W. Campbell.
E. S. Dashwood.	P. Pigott.	G. J. Wilson.

Silver Medal for Materia Medica.

W. Watson.

University of London.

Matriculation Examination.

January.	
L. F. Childe.	J. H. H. Manley.
M. M. Adler.	J. Taylor.
T. S. Jones.	
June.	
E. O. Kingsford.	J. H. Sellick.
A. Paget.	H. H. Du Boulay.

MEDALLISTS AND PRIZEMEN, 1879-80.

JULY, 1880.

The Treasurer's Gold Medal for Medicine.

Percy Warner, Fareham, Hants.

The Treasurer's Gold Medal for Surgery.

William Wenmouth Fryn, Saltash, Cornwall.

Sands Cox Scholarship.

Edwin James Wenyon, Darlington.

Third Year's Students.

George S. Mahomed, Brighton, First Prize, £35.

John Dowson, London, } (equal) £10 each.
Percy Warner, Fareham, }

Second Year's Students.

Edwin James Wenyon, Darlington, } (equal) £17 10s. each.
Sidney Worthington, Liverpool, }

Wheulton Hind (*highly commended*), Certificate.

James Henry Targett, Salisbury, Certificate.

William Thos. Frederick Davies, Swansea, Certificate.

Michael Harris Prize.

Louis Albert Dunn.

First Year's Students.

Albert Martin, Wellington, New Zealand, First Prize, £50.

Francis Barclay W. Phillips, Brighton, Second Prize, £25.

Alfred Ernest Taylor, Acton, Third Prize, £10 10s.

Charles Pope Walker, Blackheath, Certificate.

George Arthur Johnson, Worthington, Certificate.

Francis Heatherley, London, Certificate.

SEPTEMBER, 1880.

Open Scholarship in Arts.

Richard Moody Ward, Ashburton, Devon.

Open Scholarship in Science.

Henry Walter Pigeon, Clifton, Bristol.

OCTOBER, 1880.

Gurney Hoare Prize.

Edwin A. Starling, Sutton.

Pupils' Physical Society.

Session, 1880-81.

Honorary President.—Dr. WILKS.

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PRIZEMEN FOR THE SESSION, 1879-80.

To Mr. Percy Warner, £10, for his Paper on "Chronic Rheumatic Arthritis."

To Mr. G. N. Pitt, M.A., £5, for his Paper on "Temperature."

To Mr. E. A. Starling, £5, for his Essay on "Anæmia."

To Mr. W. Fowler, B.A., £5, for his Paper on "The Physiology and Histology of Secretion."

To Mr. R. J. Ryle, B.A., £5, as the Member who had distinguished himself most in the Debates of the Session.

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WINTER COURSES.

The Winter Session commences October 1st and ends March 31st.

LECTURES.

Medicine.—Dr. WILKS and Dr. PAVY.

Mondays, Wednesdays, and Fridays, at Three.

Clinical Medicine.—Dr. WILKS, Dr. PAVY, Dr. MOXON, and Dr. FAGGE.

Saturdays, at Half-past One.

Surgery.—Mr. BRYANT and Mr. DURHAM.

Tuesdays and Thursdays, at Half-past Three, and Saturdays, at a Quarter to Three.

Clinical Surgery.—Mr. BRYANT, Mr. DURHAM, Mr. HOWSE, and Mr. DAVIES-COLLEY.

Wednesdays, at Half-past One.

Anatomy, Descriptive and Surgical.—Mr. HOWSE and Mr. DAVIES-COLLEY.

Tuesdays, Wednesdays, Thursdays, and Fridays, at Nine.

Physiology and General Anatomy.—Dr. PYE-SMITH.

Mondays, Wednesdays, and Fridays, at a Quarter-past Four.

Clinical Lectures on Midwifery and Diseases of Women.—Dr. BRAXTON HICKS.

Wednesdays, at Half-past One.

Chemistry.—Dr. DEBUS and Dr. STEVENSON.

Tuesdays, Thursdays, and Saturdays, at Eleven.

Experimental Physics.—Prof. A. W. REINOLD.

Mondays and Wednesdays, at Eleven.

Comparative Anatomy and Zoology.—Dr. BRAILEY.

Tuesdays and Thursdays, at a Quarter-past Two.

DEMONSTRATIONS.

Practical Surgery.—Mr. LUCAS.

Practical Anatomy.—Dr. R. E. CARRINGTON, Dr. P. HORROCKS, and Dr. W. H. WHITE, *Demonstrators.*

And two Assistant Demonstrators.

Morbid Anatomy.—Dr. FAGGE and Dr. GOODHART.

Daily, at Half-past Two, throughout the year.

Cutaneous Diseases.—Dr. PYE-SMITH.

Tuesdays, at Twelve, throughout the year.

Practical Physiology.—Mr. GOLDING-BIRD.

Mondays and Saturdays, at Ten, Wednesdays, at One.

Practical Pharmacy.—Throughout the year.

SUMMER COURSES.

The Summer Session begins May 1st and ends July 31st.

LECTURES.

Materia Medica and Therapeutics.—Dr. MOXON.

Tuesdays, Thursdays, and Fridays, at Three.

Midwifery and Diseases of Women.—Dr. BRAXTON HICKS and Dr. GALABIN.

Tuesdays, Wednesdays, Thursdays, and Fridays, at Nine.

Medical Jurisprudence.—Dr. STEVENSON.

Tuesdays, Thursdays, and Saturdays, at Ten.

Clinical Medicine.—Dr. PYE-SMITH, Dr. F. TAYLOR, and Dr. GOODHART.

Wednesdays, at Half-past One.

Clinical Surgery.—Mr. LUCAS, Mr. GOLDING-BIRD, and Mr. JACOBSON.

Fridays, at Half-past One.

Ophthalmic Surgery.—Mr. BADER, *Thursdays, at Two.*

Clinical Lectures on Diseases of Women.—Dr. GALABIN, *Tuesdays, at Three.*

Pathology.—Dr. FAGGE, *Saturdays, at Nine.*

Hygiene.—Dr. F. TAYLOR, *Thursdays, at a Quarter-past One.*

Mental Diseases.—Dr. SAVAGE.

Tuesdays, at Eleven, and Fridays, at Half-past Ten.

Botany.—Mr. BETTANY.

Tuesdays, Thursdays, and Saturdays, at Half-past Eleven.

Dental Surgery.—Mr. MOON.

DEMONSTRATIONS.

Practical Chemistry.—Dr. DEBUS, F.R.S.

Mondays, Wednesdays, and Fridays, Ten to One.

Operative Surgery.—Mr. LUCAS.

Mondays, Wednesdays, and Fridays, at Four.

Morbid Histology.—Mr. JACOBSON, three days in the week.

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The House Surgeons and House Physicians, the Obstetric Residents, Clinical Assistants, and Dressers are selected from the Students according to merit, and without payment. There are also a large number of Junior Appointments, every part of the Hospital practice being systematically employed for instruction.

SCHOLARSHIPS AND PRIZES.

Entrance Scholarships.—An OPEN SCHOLARSHIP in Science of the value of One Hundred and Twenty-five Guineas, tenable for one year, will be competed for on MONDAY, September 25th, 1881, and following days. The subjects of the examination are—Physics, Inorganic Chemistry, Botany, and Zoology.

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For further information apply to the Dean, Dr. F. Taylor.

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THE Condition annexed by the Testator is, “That the Essays or Treatises to be written for such Prize shall contain original experiments and observations, which shall not have been previously published, and that each Essay or Treatise shall (as far as the subject shall admit of) be illustrated by preparations and by drawings, which preparations and drawings shall be added to the Museum of Guy’s Hospital, and shall, together with the Work itself and the sole and exclusive interest therein and the copyright thereof, become henceforth the property of that Institution, and shall be relinquished and transferred as such by the successful candidate.”

And it is expressly declared in the Will “That no Physician or Surgeon, or other officer for the time being, of Guy’s Hospital or of St. Thomas’s Hospital, in the Borough of Southwark, nor any person related by blood or affinity to any such Physician or Surgeon, for the time being, or to any other Officer for the time being in either of the said Hospitals, shall at any time receive or be entitled to claim the Prize.” But, with the exception here referred to, this Prize is open for competition to the whole world.

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Each Essay or Treatise must be distinguished by a Motto, and be accompanied by a sealed envelope containing the name and address of the Writer. None of the envelopes will be opened except that which accompanies the successful Treatise. The unsuccessful Essays or Treatises, with the illustrative preparations or drawings, will remain at the Museum of Guy’s Hospital until claimed by the respective writers or their agents.

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 3. Notes of Three Cases of Injury to the Elbow, with Remarks on their Diagnosis. By G. A. Wright, M.B.
 4. Notes on the History of the Physiology of the Nervous System. Taken more especially from writers on Phrenology. By S. Wilks, M.D., F.R.S.
 5. Clinical Cases of Disease of the Brain. I. Abscess; II. Embolism; III. Tumour. By S. O. Habershon, M.D.
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- List of Pupils who have passed the Examinations of the several Universities, Colleges, &c.
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List of Pupils who have received Appointments at Guy's Hospital.

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